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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

THE AMERICAN ACADEMY OF PERIODONTOLOGY

Spring Conference

April 25, 26, 27, 1960

Ann Arbor, Michigan

1960 Annual Meeting

October 13, 14, 15, 1960

Mirimar Hotel, Santa Monica, Calif.

JAMES P. HOLLERS, D.D.S.

President, American Academy of Periodontology, 1960

Dr. James P. Hollers, practicing periodontist of 325 Burr Road, San Antonio, Texas, was installed as President of the American Academy of Periodontology in New York recently. Dr. Hollers has played an important role in many of the activities of the Academy for many years and his promotion to President is a just reward for his many contributions.

Dr. Hollers was graduated from Texas Dental College and then practiced in Louisiana for four years before moving to San Antonio, Texas. He has been very active in organized dentistry and this year is President of the Texas State Dental Association. He is a member of the American Academy of Restorative Dentistry, a Fellow of the American College of Dentists, and is consultant to the Surgeon General of the United States Air Force, the Commission on Reorganization of the Executive Branch of the government, the Council on Federal Government Dental Services. He is also a member of the Armed Forces Medical Policy Council of the Office of the Secretary of Defense, Chairman of the San Antonio Medical Foundation, and served for several years as member and President of the San Antonio Board of Education. He was recently appointed a Brigadier General in the United States Air Force Reserves.

Because of Dr. Hollers' broad experience and his understanding of organized dentistry and periodontology, he will be an outstanding President of the American Academy of Periodontology.

THE JOURNAL OF PERIODONTOLOGY

JANUARY, NINETEEN HUNDRED SIXTY

The Relationship of Certain Dietary Factors to Calculus-Like Formation in Albino Rats

by WALTER H. STEWART, M.S., D.D.S. AND GEORGE W. BURNETT, D.D.S., PH.D.,
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CALCULUS is a major oral problem, particularly in relation to periodontal disease. While the exact mechanism of calculus formation has not been defined, bacteriological, enzymatic, and physico-chemical theories have been suggested to explain the phenomenon.[1]

The bacteriological theory holds that oral microorganisms are the primary cause of calculus[2-14] and that they are involved in its attachment to the tooth surface.[3,8,15-18] Leptotrichia and actinomyces have been considered most often as the causal microorganisms,[4-9,11-13] although many other bacterial species have been observed in forming and formed calculus.[19]

The enzymatic theory of calculus formation has involved principally the action of phosphatases derived from either oral tissues or oral microorganisms on some salivary phosphate-containing complex, most likely phosphoric esters of the hexophosphoric group.[3-10,13,15,19-23] This theory has been supported by a number of investigators,[20,21] has not been substantiated by others,[24,25] and has been regarded by some as entirely untenable.[1]

The physico-chemical theory of calculus formation, which largely originated with Prinz,[26] involves factors which alter the stability of colloids and decrease the solubility of calcium salts. These factors have included stagnation of saliva[26] and the activities of carbon dioxide,[9,21,26-32] ammonia,[33-36] salivary proteins,[37] and diet.[21,38]

Several animal species have been used in attempts to elucidate the mechanism of calculus formation and to establish the specific role, if any, of the various theories in calculus formation. "Calculus," "concretions," "tartar," "plaque formation," or "gingival plaques" have been observed in Syrian hamsters,[39-46] ferrets,[47-52] and albino rats.[53,54] There is no general agreement among the various investigators however, as to whether the oral concretions are similar in different animal species or if the animal concretions are similar to human calculus.[39-54] Some of the existing disparity arises perhaps from the lack of a precise definition of "calculus." Regardless of whether the plaques seen in several susceptible

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animal species are similar to or identical with human calculus, it is evident that attempts to produce a human-type of calculus in susceptible animals should be continued in order to develop suitable methods for studying the factors involved in calculus formation. Since diet has been indicated as a possible factor in plaque formation and the accumulation of concretions in previous animal studies,[45,46] an investigation was made of the effect of several dietary factors on calculus-like formation in caries-resistant Holtzmann-Rolfmeyer rats of a strain used in previous studies by Stephan and Harris.[54] The dietary factors investigated in the present study were an increase in the protein content of the diet by the addition of dried blood serum (Loeffler's dehydrated blood serum, Difco) and peanuts, alterations of the Ca:P ratio, and addition of $NaHCO_3$.

EXPERIMENTAL PROCEDURES

The diets used in the various experiments were as follows:

1. A standard laboratory checkers diet.
2. Diet 580:[54]

32 per cent skim milk powder.

66 per cent granulated cane sugar.

2 per cent Wilson's whole dried liver substance.

3. Basic Calculus Diet:

	gm.
Granulated cane sugar	185
Peanut powder	150
Gelatin	150
Agar	150
Cornstarch	100
Loeffler's dehydrated blood serum (Difco)	100
Brewer's yeast	50
Alfalfa meal	50
Oil (Cottonseed)	20
Modified Day Ca-and P-free Salt Mixture*	20
Salt Additives I, II, or III	25 or 65
TOTAL	1,000 or 1,050

SALT ADDITIVES FOR MODIFICATION OF THE BASIC CALCULUS DIET

Salt Additives in gm. per 975 gm. of the Basic Calculus Diet

	I	II	III
KH_2PO_4	12.5	7.0	12.5
$CaCO_3$	12.5	18.0	12.5
$NaHCO_3$			40.0
Approximate Ca:P Ratio	2:1	5:1	2:1

Three modifications of the salt mixtures were added to the basic calculus diet giving calculus diets I, II, and III. Diet I had a Ca:P ratio of approximately 2:1, diet II had a Ca:P ratio of approximately 5:1, and diet III had a Ca:P ratio of approximately 2:1 and approximately 4 per cent $NaHCO_3$. The animal used in the various experiments was a National Institute of Health strain of Holtzmann-Rolfmeyer Norway rat used previously by Stephan and Harris[54] to study the development of smooth surface caries, utilizing diet 580. Twenty-five day-old male and female animals were placed on the various dietary regimens. In the first group of 20 animals, 16 were placed on diet 580, and 4 which served as control for the 16 were placed on a standard checkers diet. This entire group of animals actually served, in turn, as control for the other groups of animals which were placed on the three modifications of the calculus-producing diet. Three groups of 25 day-old animals, each group consisting of 10 males and 10 females were placed on calculus diets I, II, and III. The animals were segregated as to males and females and maintained, two in a cage. During the experimental period, all animals were maintained on the particular diet and water, *ad*

*Modified Day Ca-and P-free Salt Mixture.[54(a)]

	gm.
NaCl (Morton's Iodized)	398.8
KCl	182.0
$KHCO_3$	262.0
$MgSO_4$ (Anhydrous)	120.0
$CuSO_4$ "	4.0
$MnSO_4$ "	2.0
$ZnSO_4$ "	6.0
$FeCl_3 \cdot 3H_2O$	25.0
K_2SO_4 (Anhydrous)	0.2

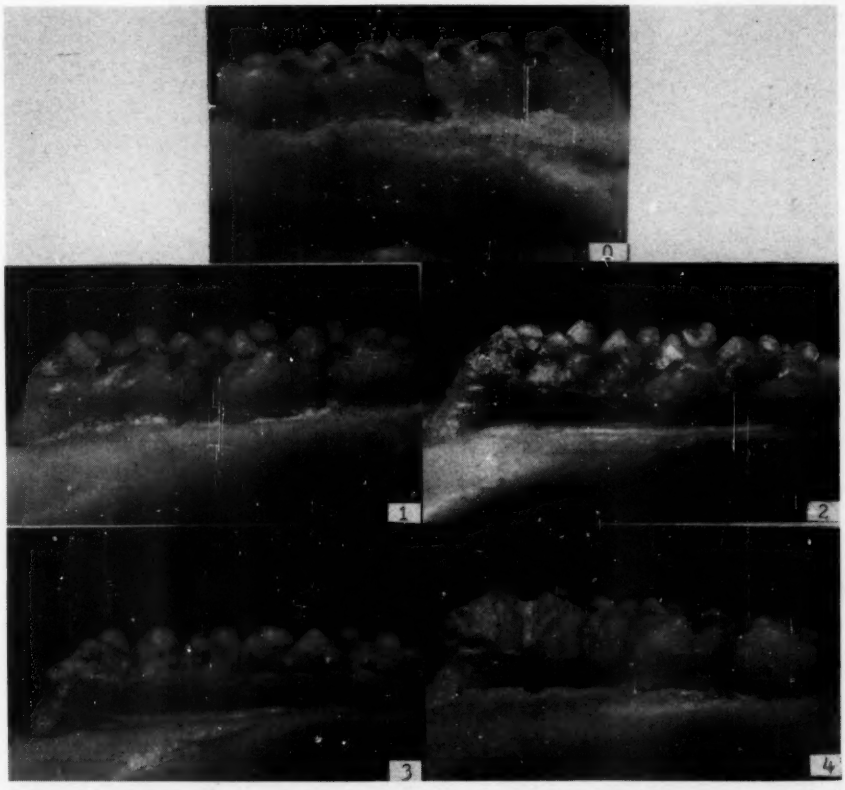


Fig. 1. Diagrammatic representation of calculus-like deposits or concretions on the upper first molar teeth of the rat with the scores in each instance as follows: 0 = No calculus-like deposit or concretions. 1 = Light calculus-like deposits occurring supragingivally. 2 = Continuous calculus-like deposits on the mesial surface of the upper first molar. 3 = Heavy calculus-like deposits on the mesial and buccal surfaces of the upper first molar, sometimes extending to the upper second molar. 4 = Heavy calculus-like deposits on mesial surface of upper first molar forming a shelf over the gingival crest, sometimes extending to the upper second molar.

libitum and at an environmental temperature of approximately 75°F. Each animal was weighed at two week intervals and the oral condition observed by means of a nasal speculum. During the week preceding the time when all the animals were sacrificed, three lactobacilli counts were made on alternate days on each animal, according to a modification of the method of Rosen, Ragheb, Hoppert, and Hunt.[55] Their procedures were followed exactly, except that after being suitably diluted in normal saline, each sample was cultured in a poured plate of Rogosa SL agar (Difco) instead of being cultured on the surface of the agar, according to the original method.

At the time of sacrifice, the animals were given an overdose of chloroform and decapitated. Color and black and white photographs were made of all oral cavities, after which the heads were autoclaved, cleansed of soft tissue so as not to unduly disturb the calculus-like deposits on the molar teeth, and dried. The caries score of each animal was determined according to the method of Cox.[56] Calculus appeared to form predominately on the mesial and buccal surfaces of the maxillary first molar teeth. Since the amount of calculus differed from rat to rat, it was estimated under 9X

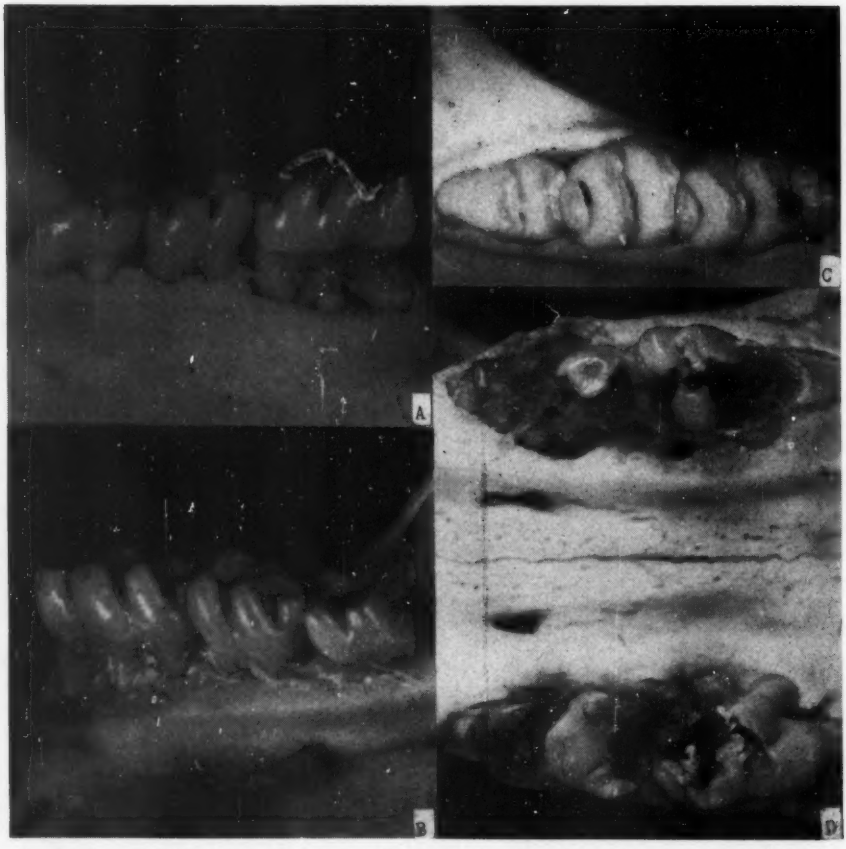


Fig. 2. Representative types of carious activity of animals on diet 580. Cemental decalcification occurred in most animals but varied in amount as in (A) and (B). Fissure caries usually began in the mesial or first fissure of the mandibular first molar (C). After 280 days on the diet there was extensive destruction of both maxillary and mandibular molar teeth (D).

magnification of a binocular dissecting microscope by a five-point grading system as is shown in Figure 1. By this system the scoring was as follows:

0 = No calculus-like deposit or concretion.

1 = Light calculus-like deposits occurring supragingivally.

2 = Continuous calculus-like deposits on the mesial surface of the upper first molar.

3 = Heavy calculus-like deposits on the mesial and buccal surfaces of the upper first molar, sometimes extending to the upper second molar.

4 = Heavy calculus-like deposits on mesial surface of upper first molar forming a shelf over the gingival crest, sometimes extending to the upper second molar.

In a particular rat, the calculus-like deposits were usually similar on either the upper right or upper left sides. The score recorded in any instance was based on the maximum amount of deposit seen on either

TABLE I

Comparison of the Calculus Status and Other Reactions of Albino Rats Maintained on Several Diets

Diet	*Sex*	*Number of Animals*	*Days on Diet*	*Weight* Gain (gm/day)*	*Fissure Caries*	*Smooth Surface Caries*	*Calculus* Scores*	*Mean Lactobacillus Counts*	*Range Lactobacillus Counts*
580	M	8	96	1.33±.11	+	+	0.75±.45	No data	100-22,000
	F	8	96	0.75±.02	+	+	1.50±.64	No data	0- 1,000
Control	M	2	280	2.63	−	−	0	0	0
	F	2	280	0.94	−	−	0	0	0
I	M	10	101	2.26±.18	−	−	2.00±.55	918± 835	3- 4,256
	F	10	101	1.51±.09	−	−	2.66±.54	34± 10	3- 68
II	M	10	102	2.60±.17	−	−	1.00±.56	409± 229	23- 1,445
	F	10	102	1.49±.06	−	−	2.00±.34	332± 121	0- 1,096
III	M	10	102	2.09±.12	−	−	3.00±.55	2,081±1,156	20- 6,150
	F	10	102	1.26±.07	−	−	2.55±.41	241± 152	3- 1,433

*Mean and Standard error of mean except for control animals.

the right or left maxillary molars. Mandibular molars were not considered in computing the scores since calculus-like deposits were not observed about these teeth, except in one animal.

RESULTS

During the experimental period some of the animals were observed periodically, especially those on diet 580, to ascertain the progressive effect of the diets on calculus formation. In general, the groups of animals were maintained on the diets for 96 to 102 days before sacrificing, except for the small control group of 4 animals which were maintained on the checkers diet for 280 days. The results of the various experiments are summarized in Table I.

The mean weight (in grams gained per day) is shown in Table I, and compared statistically in Table II. Significant and nonsignificant differences in weight grain were determined by the standard error of the difference of means. Since the number of animals involved is small only the highest significance at the 1 per cent level is recorded as significant. The weight gain of the animals on diet 580 differed significantly from the weight gains of all the other groups of animals. The weight gains of the animals on the checkers diet differed significantly from those of the other groups except for the animals on calculus diet I and the males on calculus diet II. The weight gains of animals on calculus diet I (Ca:P ratio, 2:1) differed significantly only from those of diet 580 and the females on diet III. The weight gains of animals on diet II (Ca:P ratio, 5:1) differed signifi-

TABLE II

Statistical Comparisons of the Weight Gains of Albino Rats Maintained on Several Diets

		Diet				
Diet	*Sex*	*580*	*Control*	*I*	*II*	*III*
580	M		+	+	+	+
	F		+	+	+	+
I	M	+	−		−	−
	F	+	−		+	−
II	M	+	−	−		+
	F	+	+	−		+
III	M	+	+	−	+	
	F	+	+	+	+	

+=1 per cent significance by the method of standard error of the difference of means.

−=Not significant at 1 per cent by the method of standard error of difference of means.

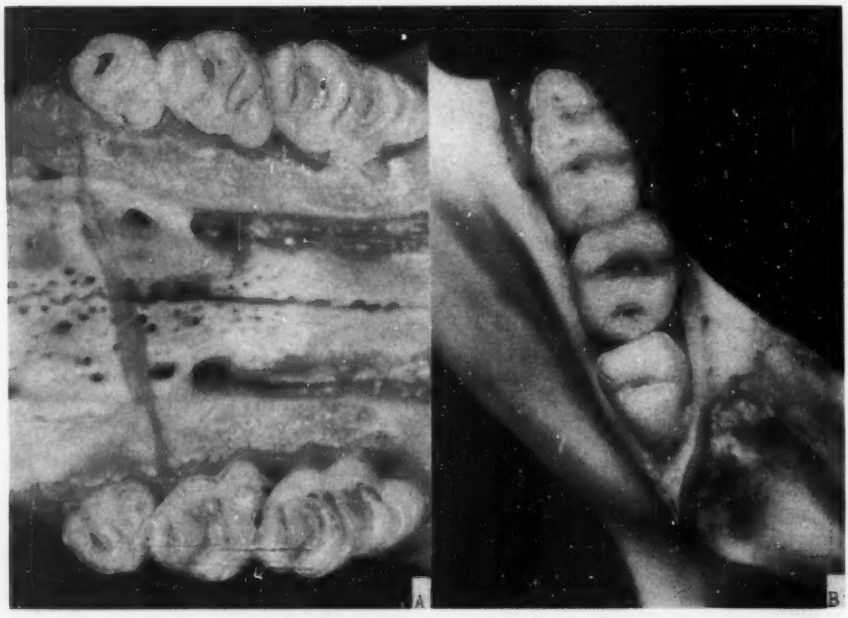


Fig. 3. Representative maxilla (A) and mandible (B) of animals maintained on calculus diets I, II, and III, showing lack of carious activity. There is some discoloration and accumulation of material in the sulci but no evidence of carious destruction.

cantly from those of all other animals except males on diet II. With group III animals (Ca:P ratio, 2:1, 4 per cent $NaHCO_3$), the weight gains differed significantly from all other animals except for those on calculus diet I.

Caries incidence was determined on all animals by the method of Cox.[56] Both fissure and smooth surface caries occurred only in the animals on diet 580 (Fig. 2). Cemental decalcification, related to soft plaque formation, occurred in most of the animals but the amount of decalcification differed from animal to animal (Fig. 2A and 2B). Fissure caries which occurred in animals on diet 580 usually began in the first fissure of the mandibular first molar (Fig. 2C) and progressed to destroy the mesial cusp. In many instances, complete destruction of both mandibular and maxillary molars occurred when the rats were maintained for 280 days on diet 580 (Fig. 2D).

Neither smooth surface nor fissure caries was observed in any of the animals maintained on either the checkers diet or on calculus diets I, II, and III, even when carefully observed with the dissecting microscope (Fig. 3). In some of these animals, debris had accumulated in the fissures and there was some discoloration but no evidence of carious destruction at the time of sacrifice. The checkers diet was sufficiently abrasive so that the molar cusps were usually worn down after some time on the diet (Fig. 3).

The first observable concretions which developed on the teeth occurred as soft, materia alba-like plaques on the lower incisors after the animals had been on the calculus diets for 21 days (Fig. 4). Later, plaques began to accumulate on maxillary first molars and in some instances, in the more severe cases, to accumulate also on the maxillary second molars. Concretions did not accumulate on the mandibular molars,

Fig. 4. Initial development of soft, materia-alba-like plaques about the lower central incisors of animals on the calculus diets for 21 days.

except in one animal on calculus diet III. As the animals were continued on the calculus diets, the soft plaques began to calcify and harden so that by the time the animals were sacrificed, the calculus-like deposits had the appearance of the usual forms of calcified human calculus (Fig. 5).

After the soft tissues were removed from the skulls, the calculus-like deposits remained tenaciously attached to the teeth. Typical example of the calculus remaining on the cleansed skulls of animals on calculus diets I, II, and III, are seen in Fig. 6. Calculus-like deposits also occurred about the maxillary teeth of the animals on diet 580 (Fig. 7), resembling that seen in animals on the calculus diets, except for the small amount and lack of calcification.

The calculus scores of the various groups of animals are compiled in Table I and their significance compared statistically in Table III according to the method of the standard error of the difference of means. No calculus was observed in the animals on the checkers diet even after 280 days. The highest mean calculus score (3.00 ± .55) was observed in the males on diet III, which had a Ca:P ratio of 5:1, while the males on diet I (Ca:P ratio, 2:1) had the lowest mean calculus score (1.00 ± .56). When comparisons were made of the mean calculus scores (Table III), significant differences were found between the females on diet 580 and checkers diet, between the males on diet 580 and calculus diet II, and between all animals on diet 580 and calculus diet III. The calculus scores of both males and females on the checkers diet differed significantly from similar animals on calculus diets I and II, while only females on the checkers diet differed significantly from similar animals on diet 580 and calculus diet II. No significant differences were found between the calculus scores of animals on calculus diet I (Ca:P ratio, 2:1) and animals on any of the other diets. The calculus scores of male animals on calculus diet II (Ca:P ratio, 5:1) differed significantly only from those of male animals on diet 580 and calculus diet III. Both male and female rats on calculus diet III (Ca:P ratio, 2:1, 4 per cent $NaHCO_3$) differed significantly from those on diet 580, while only the males on calculus diet III differed significantly from the males on calculus diet II.

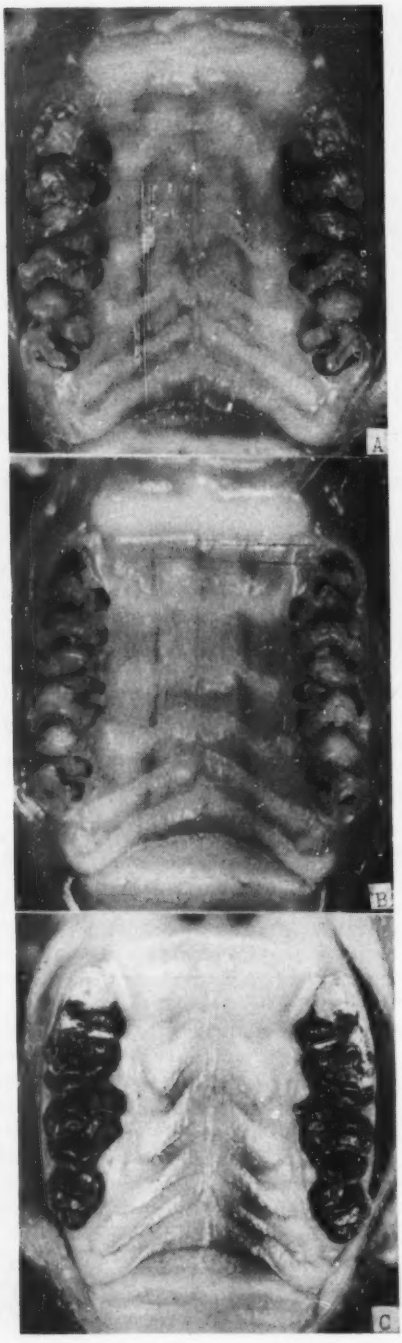
TABLE III

Statistical Comparisons of Calculus Scores of Albino Rats Maintained on Several Diets

Diet	*Sex*	*Diet*				
		580	*Control*	*I*	*II*	*III*
580	M		−	−	+	+
	F		+	−	−	+
I	M	−	+		−	−
	F	−	+		−	−
II	M	+	−	−		+
	F	−	+	−		−
III	M	+	+	−	+	
	F	+	+	−	−	

+ = 1 per cent significance by the method of standard error of difference of means.

− = Not significant at 1 per cent by the method of standard error of difference of means.



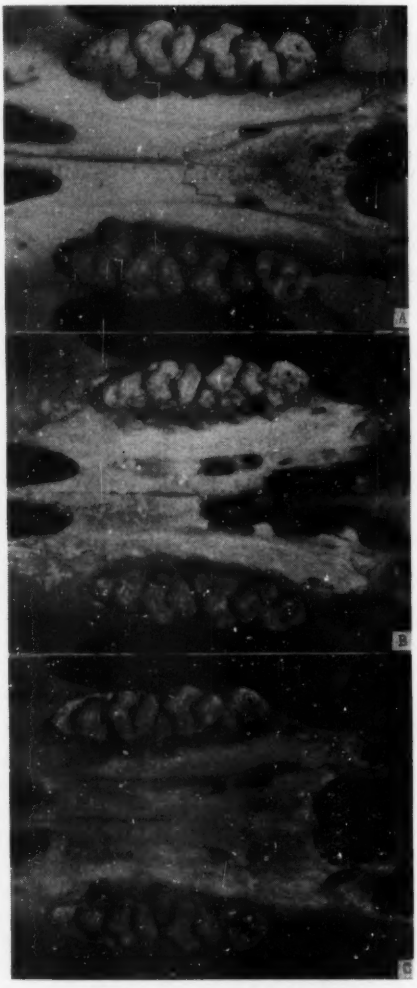


Fig. 6. Calculus-like deposits remaining on the maxillary teeth of calculus diet I animals (A), calculus diet II animals (B), and calculus diet III animals (C) after autoclaving and cleaning the skulls.

←

Fig. 5. Calculus-like deposits as they appeared in representative animals on calculus diet I (A), calculus diet II (B), and calculus diet III (C). In (C) the picture has been contrasted to show the white calculus-like deposits about the maxillary first molars which appear dark.

Fig. 7. Calculus-like deposits occurring about the maxillary first molar teeth of animals on diet 580.

TABLE IV

Statistical Comparison of the Lactobacillus Counts of Albino Rats Maintained on Several Calculus Diets

Diet	*Sex*	*Diet*		
		I	*II*	*III*
I	M		−	−
	F		+	+
II	M	−		+
	F	+		−
III	M	−	−	
	F	−	−	

+ = 1 per cent significance by the method of standard error of difference of means.

− = Not significant at 1 per cent by the method of standard error difference of means.

The results of the lactobacillus counts made during the week preceding the sacrifice of the animals are shown in Table I and compared statistically in Table IV. The lactobacillus counts were highly variable, with a significant variation occurring between the females on calculus diet I and the females on calculus diets II and III. Significant differences were found also between the females on calculus diets I and II and males on calculus diets II and III.

DISCUSSION

In calculus formation, either in man or animals, it would seem that two steps which occur are the formation of an organic or proteinaceous matrix and the deposition of an inorganic component. While these two steps could occur simultaneously, it is more likely that the formation of the proteinaceous matrix must precede that of the inorganic component for noncalcified, proteinaceous concretions are frequently observed but inorganic concretions without organic matrices have not been conclusively demonstrated. If a strictly inorganic concretion can occur initially without an organic matrix, the latter could be laid down subsequently, somewhat in the manner of secondary cuticle formation on the surface of enamel. The secondary formation of the organic matrix does not seem likely for if this situation existed, such calculus would probably contain few or no microorganisms. Quite the reverse is true, for all calcified calculus which has been observed contains an abundance of microorganisms.

In the present investigation of calculus formation, the calculus-producing diet was supplied with an abundance of protein in the form of dehydrated blood serum and peanuts on the assumption that it might either be precipitated or retained about the teeth to serve as the proteinaceous stroma for calculus, or to stimulate the growth or retention of microorganisms which might be related in some way to the initiation and formation of the organic matrix. With such a diet, a proteinaceous matrix is certainly formed within a relatively short period of time. From the data obtained in the present experiments, it has not been demonstrated conclusively, however, that the blood serum mediates the deposition of the proteinaceous matrix, although it would seem to accelerate it. Rats on diet 580, a caries-producing diet containing 2 per cent liver substance, also developed an organic matrix, although not so rapidly as did the animals on the calculus diet containing dehydrated blood serum and peanuts. Other investigators have observed the formation of proteinaceous plaques about the teeth

of ferrets and hamsters when maintained on certain caries-producing diets which did not contain a large amount of protein.[39,42-46,48,52] In most such experiments and in some of our Holtzmann-Rolfmeyer rats on diet 580, such proteinaceous matrices apparently did not progress to the degree of calcification for them unequivocally to be described as "true," hard calculus. Rather, the concretions have remained uncalcified, and have produced a distinctive type of cervical decalcification of dentin (Fig. 1A, 1B). In some instances the proteinaceous stromas also have produced periodontal disturbances. When our animals were maintained for sufficient time on caries-producing diet 580, caries progressively destroyed the teeth, in most instances (Fig. 1C, 1D).

In contrast, the proteinaceous matrices of plaques about the teeth of the animals on the calculus diets (which were essentially not conducive to caries activity) proceeded to a degree of calcification indistinguishable clinically, at least, from ordinary, hard calculus such as is seen in human beings. It was our observation that calculus diet III, which had a Ca:P ratio of 2:1 and a $NaHCO_3$ content of 4 per cent, was most conducive to calcification, as judged by the amount of calculus-like material which accumulated. Animals on the non-caries-producing diets with a Ca:P ratio of 2:1 and 5:1 also accumulated considerable of the calculus-like material.

Undoubtedly, carbon dioxide and carbonates greatly influence saliva.[57-60] It has been postulated that the carbon dioxide content of the blood controls the carbon dioxide content of saliva. The carbonate of the saliva is believed to be a principal buffer and the amount to inversely influence pH, i.e., if the carbon dioxide increases then the pH is more acid and vice versa. It has been stated that the intake of sodium bicarbonate increases the carbon dioxide content of blood and saliva, and hence the pH of saliva decreases when sodium bicarbonate is ingested. Based on such observations, the sodium bicarbonate content of calculus diet III was increased to approximately 4 per cent to determine its effect, if any, on calculus formation in the presence of a non-caries-producing diet, with a Ca:P ratio of 2:1. For whatever reason, animals on the diet accumulated more calculus on an average than did the animals on diets containing only calcium and phosphorous in a 2:1 or 5:1 ratio.

Some differences in the response of males and females to the calculus diets were observed. Such observations in regard to calculus formation certainly should be investigated further as well as many other aspects of the mechanism of calculus formation in regard to diet.

SUMMARY

An investigation has been made into the effect of increasing the calculus-like formation in a caries resistant strain of Holtzmann-Rolfmeyer rats. Increasing the protein content of the diet by addition of Loeffler's dehydrated blood serum and peanuts, altering the calcium: phosphorous ratio, and the addition of sodium bicarbonate to the diet of rats increased the rate of formation and the amount of calculus in comparison to the amount of calculus accumulating in similar animals on laboratory chow or on a caries-inducing diet (diet 580). Animals on the high protein diet and normal or altered calcium: phosphorous ratio formed plaques on the mesial and buccal surfaces of maxillary first molar teeth which were eventually fully calcified, similar to hard human calculus. Animals on the calculus diet plus 4 per cent sodium bicarbonate formed the most calculus. Neither plaques nor calculus formed on mandibular molars, except in one instance. Some differences were noted between calculus formation in males and females. Animals on laboratory chow formed no concretions or plaques on the teeth. Animals on a caries-inducing diet (580) formed a small amount of concretions about the molar teeth which did not fully calcify.

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Failure of Human Saliva to Form Calculus on Rat Molars

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WHAT role do microorganisms play in the formation of calculus in the human mouth? Microorganisms are present in calculus (2, 9). Experiments have shown that human saliva will form calculus in vitro if it contains live organisms, but will not form calculus if the organisms have been destroyed (12, 20, 21, 27, 32).

Albino rats do not usually have dental calculus (19). Would the continuous introduction of human saliva into the rat's mouth cause calculus to form (4)? This study was designed to answer that question.

MATERIALS AND METHODS

Saliva was collected from dental students who were known calculus formers, and it was immediately quick-frozen for storage. Examination of the frozen saliva indicated it was similar bacteriologically to the fresh saliva.

Twenty, 150-day-old rats were used in this study: 10 experimental, 10 control.** Two soft diets were prepared. The experimental diet consisted of two parts Purina Checkers, two parts water, and three parts human saliva. The control group was fed a diet of two parts Purina Checkers and five parts water. Both diets had the consistency of thick soup. The ration was always greater than the rat's ability to consume it.

Morning and evening the feeding dishes were washed, and fresh portions of the diets were furnished. Drinking water was available at all times. Both groups stayed on their respective diets for 90 days.

Twice daily the rats were handled, and their physical condition was observed. Weight changes were recorded once a week. Each week some of the rats were anesthetized, and the oral pH was measured with pHydrion paper. They also were examined for gingival lesions, stain, and calculus.

After 90 days all rats were sacrificed, and each quadrant of the mouth was rapidly separated and fixed in 10 per cent formalin. The quadrants were critically examined under a dissecting microscope. The specimens were then processed for histologic examination, and stained with Hematoxylin-Eosin or Gram stain. In two rats serial sections of the teeth were studied. In the remainder the upper right first molar was selected as representative. Forty teeth were examined.

RESULTS

The rats ate the food without hesitation. They were docile and showed no outward signs of stress (31, 38). Their weight curves and fur were normal. The oral mucosa showed no clinical signs of inflammation. The teeth did not accumulate exogenous stain or deposits, or develop caries. The incisors retained the normal yellow color. Their oral pH remained at 9, normal for rats.

After necropsy, the teeth were examined under low magnification, and a hair line of brown stain was found at the level of the free gingival margin on two of the control rats. This was not seen at later histologic examination.

Histologic examination showed baso-

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**The experiments were conducted according to the "Rules Regarding Animal Care," as established by the American Medical Association.

This paper is based on the thesis presented in partial fulfillment of the requirements for the degree of Master of Science, Ohio State University, Columbus, Ohio, awarded December 19, 1958.

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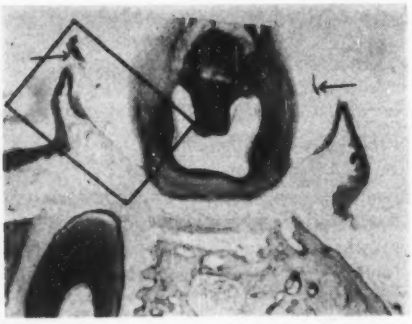

Fig. 1. Bucco-lingual section of maxillary first molar of a rat on the experimental diet. Arrows point to plaque. Mag. 40X.

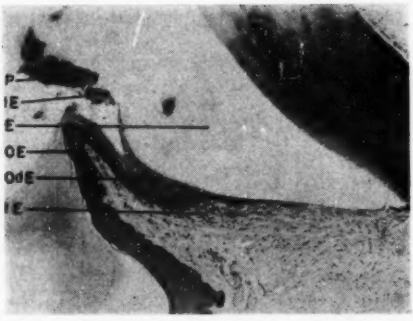

Fig. 2. Higher magnification of area in fig. 1. box. (E) Enamel. (OE) Oral Epithelium. (OdE) Odontogenic epithelium. (P) Plaque. Note laminations. (IE) Inflammatory cells. Mag. 160X.

philic foreign material, colonies of microorganisms, and degenerating cells on 9 teeth (Fig. 1). These accumulations were designated plaque (26). In the experimental group, 6 had none, 2 had slight, and 2 had moderate plaque formation. In the control group, 5 had none, 4 had slight, and 1 had moderate plaque. Therefore, it was felt the presence of human saliva in the rat mouth did not cause plaque to form.

Gram stains of representative decalcified sections showed that yeasts, and gram positive rods and cocci were common in the plaques of both experimental and control rats. The experimental rats, had, in addition, isolated colonies of gram positive streptococci, diplococci, and curved rods, and a moderate amount of actinomyces. From this we could infer that, although the human microorganisms were present in the rats' mouth, other conditions are essential to the formation of calculus.

There was some proliferation and downgrowth of sulcus epithelium in both groups; this resembled the normal aging process of the albino rat (3, 34, 41, 45). There apparently was no relation between this process and the presence of plaque or other visible irritants.

DISCUSSION

In this study the rats' mouths were inoculated with all the elements which are presumed to be necessary to form calculus (11, 30, 44). Local factors, such as high pH (10, 15, 16, 29, 36, 37) and soft diet (17, 28), should then favor precipitation of calcium salts. Plaque formation in rats fed human saliva was the same as in those fed a control diet.

A reappraisal of the processes of mineralization and calcification in other parts of the body might shed some light upon the apparent failure of the plaque to calcify in this study.

The complex carbonate hydroxy apatite crystal is the principal mineral component of bone and teeth, and of most pathologic calculi (22). This crystal is always found intimately associated with organic material which apparently acts as a matrix. It is generally accepted that this organic matrix precedes the deposition of mineral substances (5, 14). The property which permits or induces the deposition of minerals is presumed to depend upon the spatial configuration and the chemical bonding potential of the surface of the organic matrix micelles (1, 8, 33, 35, 43, 46). Local factors could effect the rate of mineralization (42).

In bone matrix, collagen fibrils seem to

have the property of calcifiability. In pathologic calcifications the calcifiable matrix may be made up in part of bacterial cell bodies and their exudates (6, 13, 26, 50). The bacteria may alter the tissue exudates, or desquamated epithelial cells, which become the calcifiable matrix (24, 25).

In this study there always were cells between the plaque and the margin of the gingiva (Fig. 2). The orientation of the laminations of the plaque (Fig. 2) seems to suggest the possibility that these cells contribute to the plaque (47, 48, 49).

The plaque, when it did form under the conditions of this experiment, probably did not acquire calcifiability. This needs further study. It may be that the intensity of inflammation affects the calcifiability of the matrix (7, 18, 39, 40).

CONCLUSION

Human saliva was mixed with rats' food to make a mushy pabulum. This was fed to ten rats morning and evening for 90 days. During this time no calculus formed. Occasionally some plaque formed. Since ten rats on a control diet showed similar results, it was felt that the presence of human saliva was not significant.

The plaque that formed seemed to be related to the surrounding tissues in a definite manner. This suggests the possibility that the gingival tissue exudates may be a factor in plaque formation, and supports the concept of apatite crystal formation in a template matrix.

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Healing of Human Periodontal Tissues Following Surgical Detachment and Extirpation of Vital Pulps

by MELVIN L. MORRIS, B.S., M.A., D.D.S., *New York, N. Y.*

STUDIES of surgically created periodontal pockets[1,2] have demonstrated healing directly with the dentin of vital teeth and cementum of vital and non-vital teeth.

Healing did not occur directly with the dentin of teeth with root canal fillings. When this exposed dentin was connected with the gingival crevice the epithelial lining grew apically until the first cementum was encountered. Denuded dentin remote from the gingival crevice showed a very slight amount of new cementum growing in from the surrounding cementum in an incomplete attempt to cover the defect.

Several teeth with gangrenous pulps reacted like root-canal filled teeth where exposed dentin was connected with the gingival crevice but showed profuse cemental deposition (as in vital teeth) on the dentin remote from the gingival crevice. This confirmed a previous observation by Waerhaug.[4,5]

Many questions remain concerning the effects of non-vitality on periodontal wound healing. It is not certain whether pulpal death alone inhibits a connective tissue union with the exposed dentin or whether the additional complication of a root canal filling and its medications are necessary for this effect. It is also possible that residual areas of functioning odontoblasts may be responsible for the new cemental formation in gangrenous teeth.

To answer some of these questions it was decided to repeat the experimental wounds on teeth whose vital pulp contents had previously been mechanically removed without use of either chemicals or root canal fillings of any kind.

PROCEDURE

Pulp contents were completely removed with broaches, reamers, and files, both ends of the canals being left open. One week later the surgical periodontal pockets were created, removing alveolar bone when necessary to deepen the wound. The free gingival rim was removed to delay epithelial invagination. Dentin was exposed at the apical end of surgical separation, and in the coronal half of the wound. In some cases cementum was curetted. Additional tooth nicks were formed as landmarks at the original crevice depth and on the enamel. Measurements were recorded from enamel nick to gingival margin and gingival attachment before, immediately after creation of the pocket, and at the time of the biopsy. This information made it possible

TABLE I

Case no.	*MM Surgical detachment*	*MM Reattachment*	*Cementum curetted*	*Alveolar bone removed*
1	4.0	3.0	No	No
5	3.5	1.5	No	Yes
6	5.0	4.0	No	No
7	3.0	3.0	No	Yes
8	4.0	3.0	No	Yes
9	6.0	4.5	No	Yes
10	4.0	2.5	Yes	Yes
11	5.0	4.5	No	Yes
12	4.0	2.0	No	Yes
13	—	2.5	Yes	No
14	6.0	1.0	Yes	Yes
15	6.0	4.5	Yes	Yes

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Part of a paper, "Studies in Periodontal Healing and Regeneration," presented at the American Academy of Periodontology, Dallas, Texas, November 7, 1958.

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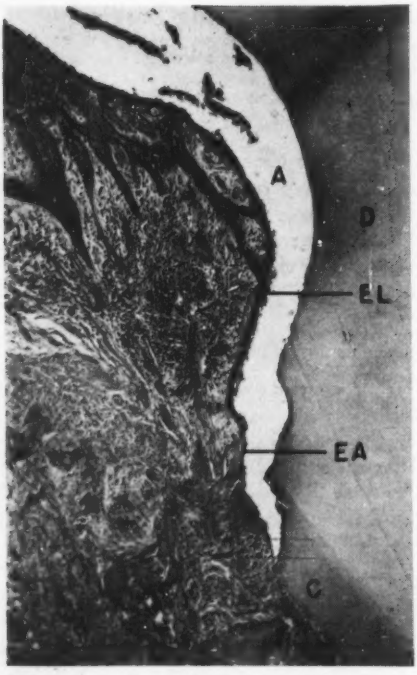

Fig. 1. The epithelial lining (EL) has grown past the denuded dentin (D), stopping at the remaining cementum (C). (A) is a separation artefact.

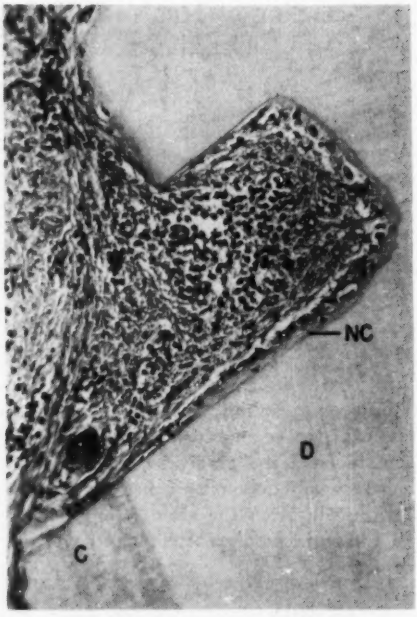

Fig. 2. New cellular cementum (NC) has formed on dentin (D) in the nick (N) located at the apical end of surgical separation. (C) is remaining cementum apical to the nick.

to determine changes in gingival attachment level with reference to a fixed point. Biopsies were performed as previously described.[1,2]

The material reported on consists of eleven maxillary anterior teeth from apparently healthy adults. Experimental periods ranged from four to seven weeks.

RESULTS

A) *Clinical:*

Table I shows that healing occurred in every case but was short of the amount of surgical detachment by from 0.5 to 5.0 millimeters except case #7 where healing equaled the amount of detachment.

B) *Histological:*

The results were consistent in that epithelial lining grew past denuded dentin connected with the gingival crevice and stopped where the first cementum was met (Fig. 1). It made no difference whether that cementum had been untouched, curetted, or partly removed with a bur.

The exposed dentin in the terminal nicks was, in the majority of cases, covered with a pink amorphous cementoid material often enclosing connective tissue cells (Fig. 2). As in previous studies[1,2,3] the new material was thickest toward the deeper portions of the nicks. Its thickness varied from .008 to .014 millimeters.

Denuded dentinal surfaces not covered by new material were marked with many resorption bays (Fig. 3) containing connective tissue cells and fibers.

DISCUSSION

The results are a curious mixture of past

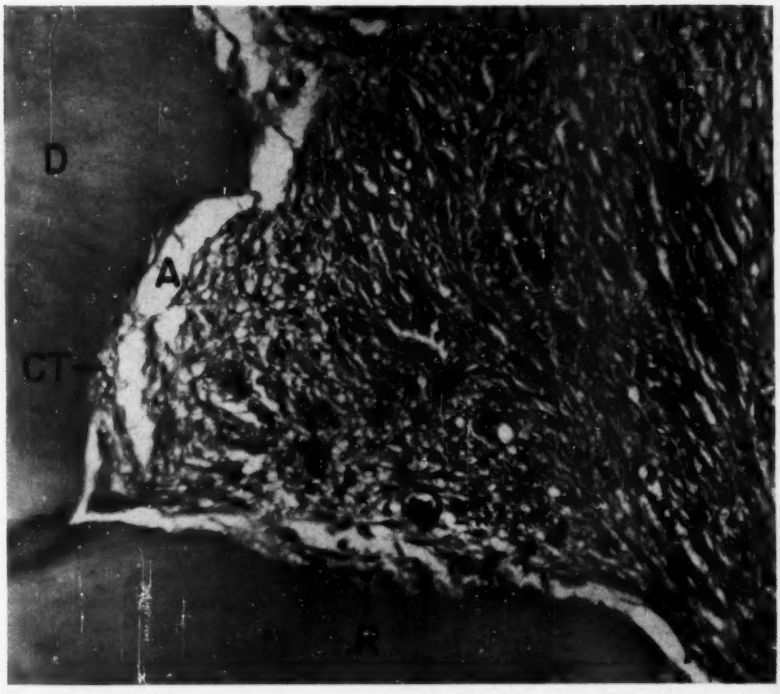

Fig. 3. Resorption bays (R) with enclosed cells are present on the apical or terminal nick (N). Connective tissue fibers (CT) are seen clinging to the dentin. (A) is a separation artefact.

studies. New cementum was deposited in the terminal nick as in vital teeth[2] but not as in root-canal filled teeth.[1] On the other hand, the epithelial lining grew down until the first cementum was encountered like the root canal filled teeth but unlike the vital teeth. No explanations or conjectures are offered at this point for the apparent contradictions.

However, the results do supply some answers to the questions that prompted this study.

It is possible that a substance (or substances) exist in dentin that encourage union with the surrounding connective tissues and induce cementoblastic activity. Although they may be elaborated in the pulp it is not necessary for a vital pulp to be present for new cementum to form on dentin. This means that new cementum could form in the presence of completely gangrenous pulps.

The absence of pulp contents *does* seem to affect the union of dentin and connective tissue in the cervical area where epithelial invagination was not stopped. This downgrowth accounts for the incomplete reattachment noted clinically.

As in the root-canal filled teeth the remaining cementum united with connective tissue and blocked epithelial downgrowth whether or not the cementum was untouched, curetted or partly removed leaving even the narrowest strip on the root where cementocytes are generally necrotic. This strengthens the idea that inductive qualities in cementum itself do not depend on its cellular vitality but reside in the intercellular substance.

Since the mere absence of pulp does not prevent cemental formation on dentin in the terminal nicks, the lack of new cementum in root canal filled teeth may have been due to the noxious influence of the medicaments employed, to the blocking of a diffusion process, or to simply a drying out of the dentin over a period of many years.

Appreciation is expressed to the many who helped to complete this study. This includes Dr. William Savoy, Dr. J. Anderson, Dr. H. Baurmash, Dr. G. Minervini, Dr. P. Vahdat and Mrs. F. Moore, Mrs. R. Feinman and Miss E. Boyd.

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WASHINGTON, D.C. GROUP ORGANIZES PERIODONTAL SOCIETY

Several diplomates of the American Board of Periodontology living in the Nation's Capital have formed a local organization known as the Greater Washington Periodontal Society. The Society's general objective will be to exchange, disseminate and evaluate data on periodontology. Its first meeting was held on November 16.

Having a current membership of thirty, the Society's eligibility rules require that members have a teaching or research appointment or have completed graduate work in the field of periodontology, and be active participants in the Society's activities. Meetings will be held about twice yearly.

Among the Society's founders were Drs. William B. Ingersoll and Francis J. Fabrizio, both of the Georgetown University Dental School; Col. Joseph L. Bernier of the Armed Forces Institute of Pathology and a former President of the American Academy of Periodontology; and Dr. Mildred Dickerson, D.D.S., of Washington.

Present officers are Dr. Paul M. Baer, of the National Institute of Dental Research, President; Maj. Charles F. Sumner, III, Walter Reed Army Medical Center, Vice President; Dr. Albert G. Paulsen, Treasurer; Lt. Cmdr. Perry C. Alexander, U. S. Naval Dental School, Bethesda, Maryland, Secretary; and Capt. Alfred L. Raphael, of the U. S. Naval Dispensary in Washington, Chairman, Membership Committee. The next meeting is to be held in April.

Studies on Periodontal Disease in the Mouse

I. EFFECT OF AGE, SEX, CAGE FACTOR, DIET

by PAUL N. BAER, D.D.S. AND CARL L. WHITE, D.D.S., *Bethesda, Maryland*

THE inbred strain of mouse, STR/N, was found to be susceptible to periodontal disease.[1] As a result, a series of experiments were designed to obtain additional information about this strain in general, and, in particular, to answer the following questions:

1. At what age does periodontal disease first manifest itself and at what minimum age can the animals be sacrificed for clear-cut observable changes useful in experimental procedures?

2. Is there a sex difference?

3. Is there a cage factor?

4. Does the physical consistency of the diet appear to be important as an etiologic factor?

5. What are the effects, if any, of high fat diets on periodontal disease (alveolar bone loss)?

6. What effects, if any, do fluorides have on periodontal disease?

METHODS AND RESULTS

Inbred mice, both male and female, of strain STR/N were used. They were all housed in plastic cages and maintained in a similar environment. At the time of sacrifice, the heads of the animals were removed, defleshed by the papain method, and scored for alveolar bone loss according to a method previously described.[1]

A. Age and Sex

Equal numbers of weanling male and female mice were divided into eight groups. The mice were housed 3-6 per cage and fed Purina Laboratory Chow. One group was sacrificed at each of the following time intervals: 30, 50, 54, 60, 75, 100, 130, and 170 days of age. A total of 162 animals were used.

The results, Table I, indicate that periodontal disease is first manifested somewhere between 30-50 days of age, and increases in both severity and incidence with age, until at 170 days of age all of the animals examined had some degree of alveolar bone loss.

No sex difference was found, (females 14.53±0.537, males 15.89±0.616).*

TABLE I

Onset of periodontal disease

Age	*Number of animals*	*Percent*
30 days	12	0
50 days	12	50
54 days	14	50
60 days	13	53
75 days	12	60
100 days	30	80
130 days	19	95
170 days	50	100

B. Cage Factor

To rule out the possible effects of a cage factor, weanling mice were housed from one to six animals per cage. A total 145 mice were used. The animals were maintained on a Purina Laboratory Chow diet and sacrificed at 170 days of age.

The results, Table II, show that there was no difference in periodontal disease whether the mice were housed one or six per cage. However, when an attempt was made to house seven or more mice per cage, deaths of some of the animals invariably occurred.

TABLE II

Analysis of variance

Source of variation	*df*	*Mean Square*	*F Ratio*
Between cages	41	10.94748	0.915
Within cages	104	11.96057	

*Standard errors.

C. Physical Consistency of Diet

Three groups of mice, 30 days of age, were used in this series. One group was fed Purina Laboratory Chow in the pellet form, the second group was fed these same pellets after they had been ground, and the third group received a synthetic diet that was believed to be similar to the commercial Purina diet. It consisted of the following:

Brewers yeast	7%
Liver powder	5%
Soy protein	18%
Alfacell	6%
Salts	6%
Sucrose	14%
Cornstarch	28%
Glucose	11%
Corn oil	5%

This synthetic diet, however, was of a much finer physical consistency than the ground pellets.

The animals were sacrificed at 170 days of age. A total of 71 animals were used.

The results, Table III, indicate that the animals on the Purina Laboratory Chow, pellet form, had the worst periodontal disease (alveolar bone loss). The mice fed ground Purina Laboratory Chow or the synthetic diet exhibited similar amounts of loss. The differences between the Purina Laboratory Chow in pellet form and the other two diets are statistically significant ($P = 0.01$).

TABLE III

Means and Standard Errors

Diet	*N*	*Mean*	*Standard Error*
Purina Lab. Chow (pellet form)	29	15.068	0.310
Ground Purina Lab. Chow	17	12.059	0.424
Synthetic Diet (700)	25	13.800	0.440

D. High Fat Diets

In this experiment, two groups of weanling mice, 62 animals in all, were used. One group was fed a diet high in a hydrogenated vegetable oil and the other high in Coconut oil. The hydrogenated vegetable oil contains a mixture of both saturated and unsaturated fatty acids, while the Coconut oil contains only saturated fatty acids (Iodine value for Coconut oil is: 7-10). The compositions of the diets used are as follows:

Diet 10,046

Hydrogenated vegetable oil (Crisco)	57%
Salts (HMW)	6%
Casein	25%
Liver powder	5%
Brewers yeast	7%

Diet 10,047

Coconut oil	57%
Casein	25%
Salts (HMW)	6%
Liver powder	5%
Brewers yeast	7%

As can be seen from Table IV, there was no difference in periodontal disease between these two groups of mice. At the time of autopsy, none of the manifestations of a fatty acid deficiency were evident in the mice maintained on diet 10,047, (57% Coconut oil).

TABLE IV

Means and Standard Errors

Diet	*N*	*Mean*	*Standard Error*
57% Hydrog. veg. oil (diet 10,046)	37	14.297	0.549
57% Coconut oil (diet 10,047)	25	12.120	1.009

E. Sodium Fluoride

Two groups of mice, 30 days of age, one fed a semi-purified diet (diet 10,046), containing 57% hydrogenated vegetable oil, and the other Purina Laboratory Chow, were given drinking water, ad libitum, which contained 60 ppm. sodium fluoride (27 ppm. F). A total of 55 experimental animals were used. The animals were sacrificed at 170 days of age. At autopsy, no signs of a fluoride toxicity was observed in

any of the animals.* Interference with incisal pigmentation mechanism was observed, however.

As can be determined from Table V, there was a statistically significant reduction in loss of alveolar bone for the animals which received water containing sodium fluoride as compared to the controls which were taking tap water.

TABLE V

Means and Standard Errors

Diet	*N*	*Mean*	*Standard Error*
Purina Lab. Chow	67	15.149	0.271
NaF + Purina Lab. Chow	27	12.037	0.422
57% hydrog. veg. oil (diet 10,046)	37	14.297	0.549
NaF + 57% hydrog. veg. oil (diet 10,046)	28	11.179	0.735

DISCUSSION

The initiation of the periodontal lesion, as indicated by loss of alveolar bone, occurred approximately with the onset of sexual maturity, and increased in severity with increase in age. These findings parallel the findings reported in human epidemiological studies by Russell[2] and Marshall-Day.[3]

Our findings that an adequate diet of a hard physical consistency affords no protection whatsoever against periodontal disease, and, as a matter of fact, in some strains of small laboratory animals actually results in an increased severity, confirms our earlier findings[1] and those of Auskaps et al.[4]

We are not able to explain the reduction in alveolar bone loss which occurred when the animals were fed ground Purina Laboratory Chow. The same is true of the preventative effects of the synthetic diet of similar chemical composition but finer physical consistency. Auskaps[4] reported similar results for the rice rats.

The lack of EFA (essential fatty acid) deficiency symptoms in the mice reared on a diet containing 57% coconut oil, an oil which contains none of the essential fatty acids, may at first seem incongruous. Aaes-Jorgensen,[5] however, described somewhat similar findings in the rat and felt it might be due to the high content of mediumchain saturated acids. These medium-chain-length fatty acids may substitute partially for the unsaturated acids in functions where similarities in physical properties permit substitution, and in this way spare the polyunsaturated acids for more vital functions.

Drinking water containing 60 ppm. sodium fluoride caused a reduction in alveolar bone loss for the mice fed a diet of either Purina Laboratory Chow or a synthetic diet containing 57% hydrogenated vegetable oil. Costich,[6] and McClenden[7] have also reported on the beneficial effects of sodium fluoride in causing a reduction of alveolar bone loss. Smaller concentrations of NaF in the drinking water in the rice rat, however, caused no apparent effects.[4]

In epidemiological studies, Russell[8] reported that there was a tendency for better periodontal health to occur in those individuals who came from communities where the domestic water supply contained fluorides as compared to those from nonfluoride areas. Our results are also in accord with these findings.

SUMMARY AND CONCLUSIONS

1. In the STR/N mouse, the onset of periodontal disease occurred between the 30-50 days of age.

2. There was no observable difference in sex nor in the number of animals housed per cage that could be related to the disease process.

3. At 170 days of age, all animals had some degree of alveolar bone loss.

4. A nutritionally adequate diet of a

*Autopsy performed by Dr. Mearl F. Stanton, National Cancer Institute.

hard physical consistency afforded no protection against alveolar bone loss.

5. A nutritionally adequate diet of a soft physical consistency, or sodium fluoride, 60 ppm. in the drinking water, resulted in a reduction of alveolar bone loss.

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Book Review . . . .

An Introduction to Periodontia by Henry M. Goldman, Saul Schluger, D. Walter Cohen, Bernard Chaikin and Lewis Fox. First Edition, 346 pages, 180 illustrations. The C. V. Mosby Co., 1959, St. Louis, Mo., price $7.75.

The statement of not judging a book by its cover has been repeated many times. In considering "An Introduction to Periodontia" one can paraphrase this remark to read one should not judge a book by its size. This pocket size edition is a well written and illustrated book with selected reading references at the end of each chapter. It was written to provide a basic background in periodontia for the student and practitioner.

The anatomy, histology and physiology of the periodontium are discussed in an understandable and uncomplicated manner in the first chapter. Following this, periodontal disturbances are classified into the conventional inflammatory, dystrophic and their combinations. Etiology is discussed in detail which is followed by examination, diagnosis, treatment planning and prognosis. All the various methods of therapy are clearly illustrated with step by step photographs showing both the instruments and procedures employed.

The book emphasizes that successful treatment of periodontal disease can be achieved only by cooperation by the patient in oral physiotherapy. Without this, all of our knowledge and skill is useless.

The writers are to be commended for such a concise report on periodontia. It will serve well as an introduction and also as a review of this field. *H. M. S.*

Further Observations Regarding the Effects of Ovariectomy Upon the Tissues of the Periodontium*, **

by IRVING GLICKMAN AND JULIAN QUINTARELLI, *Boston, Massachusetts*

IN a previous study[1] we reported that ovariectomy in 4-6 week old albino mice resulted in osteoporosis of alveolar bone, diminution in bone formative activity adjacent to the periodontal membrane and reduction in the fiber density of the periodontal membrane. No changes in the periodontium followed ovariectomy performed in one year old mice. The experimental period was sixteen weeks. As a follow-up to this initial experiment, we were interested in determining the following: the length of time required following ovariectomy for changes to occur in the periodontium; whether the periodontal changes persisted or underwent repair; whether the adrenal glands underwent changes in an attempt to compensate for the estrogen insufficiency created by ovariectomy.

EXPERIMENTAL PROCEDURE

Two age groups of female virgin albino mice RAP strain were used. The first group consisted of 134 animals, 4-6 weeks old; the second consisted of 126 one year old animals. The animals in each age group were subdivided into ovariectomized, sham-operated and control sub-groups (Chart I).

Technic of Ovariectomy and Sham Operation. Two separate costolumbar incisions were made and the ovaries were completely exposed and removed in toto, together with a part of the oviduct in order to ensure that no remnants were left. Both oviducts were tied off before removal of the ovaries. The wound was closed by suturing the muscles and the skin separately. The ovaries, along with the attached part of the oviduct, were prepared for microscopic examination to check the thoroughness of the operation. The technic for the sham operation was the same as the ovariectomy except that the ovaries and oviducts were not disturbed. Littermates were used for controls.

Animals in each sub-group were sacrificed at the following post-operative intervals; one week, two weeks, one month, two months, three months and six months (Chart I). At the time of sacrifice, the mandible, vagina and adrenals were removed and prepared in serial section for microscopic study.

OBSERVATIONS

In the vagina, at all age levels, in the control and sham-operated animals the resting phase (diestrus), proestrus, estrus or metestrus could be identified depending upon the stage of estrus at time of sacrifice (Fig. 1). In the ovariectomized animals, the vaginal epithelium consisted of only 2 or 3 layers of cells (anestrus) without signs of proliferation or other modification (Fig. 2) which might be interpreted as a recurrence of physiologic cyclical activity.

The adrenal glands of the ovariectomized animals presented no gross or microscopic differences from those of the control or sham-operated animals.

The periodontium of the animals ovariectomized at 4-6 weeks of age differed from that of the control and sham-operated animals. In the periodontal membrane, the cells were reduced in number. Differentiation of the fibroblasts to form osteoblasts and cementoblasts was impaired so that the latter cells were reduced in num-

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TABLE I
The effect of ovariectomy upon the periodontium

EXPERIMENTAL ANIMALS						
Post Oper. Interval	4—6 Weeks Old			1 Year Old		
	Control	Sham Operation	Ovariectomy	Control	Sham Operation	Ovariectomy
1 week	7	5	10	5	5	7
2 weeks	6	5	10	3	5	5
1 month	6	4	5	4	4	6
2 months	5	5	6	7	5	10
3 months	5	5	10	5	5	10
4 months	5	5	10	5	5	10
6 months	5	5	10	5	5	10
	39	34	61	34	34	58
	TOTAL 134			TOTAL 126		

ber. The regular arrangement of the collagen bundles was disorganized. The density of collagen bundles per unit area was reduced. The individual fibers were thinned and filamentous in appearance (Fig. 3).

Bone formation adjacent to the periodontal membrane was diminished or absent. There was little evidence of osteoid on bone surfaces where it normally occurred. Instead of numerous well formed osteoblasts only scattered, poorly differentiated cells were observed lining the bone surfaces (Fig. 3).

Cementum formation was impaired in the ovariectomized animals. Instead of a well formed border of cementoid with adjacent cementoblasts the experimental animals presented thin granular surface accumulations of partly calcified cementum and poorly formed cementoblasts. Impairment in cementum formation became apparent 3 months following ovariectomy, but there was no difference in cemental thickness in the experimental and control animals at this stage. However, by the sixth post-operative month the cementum of the ovariectomized animals was only half as thick as that in the control and sham-operated animals (Fig. 3).

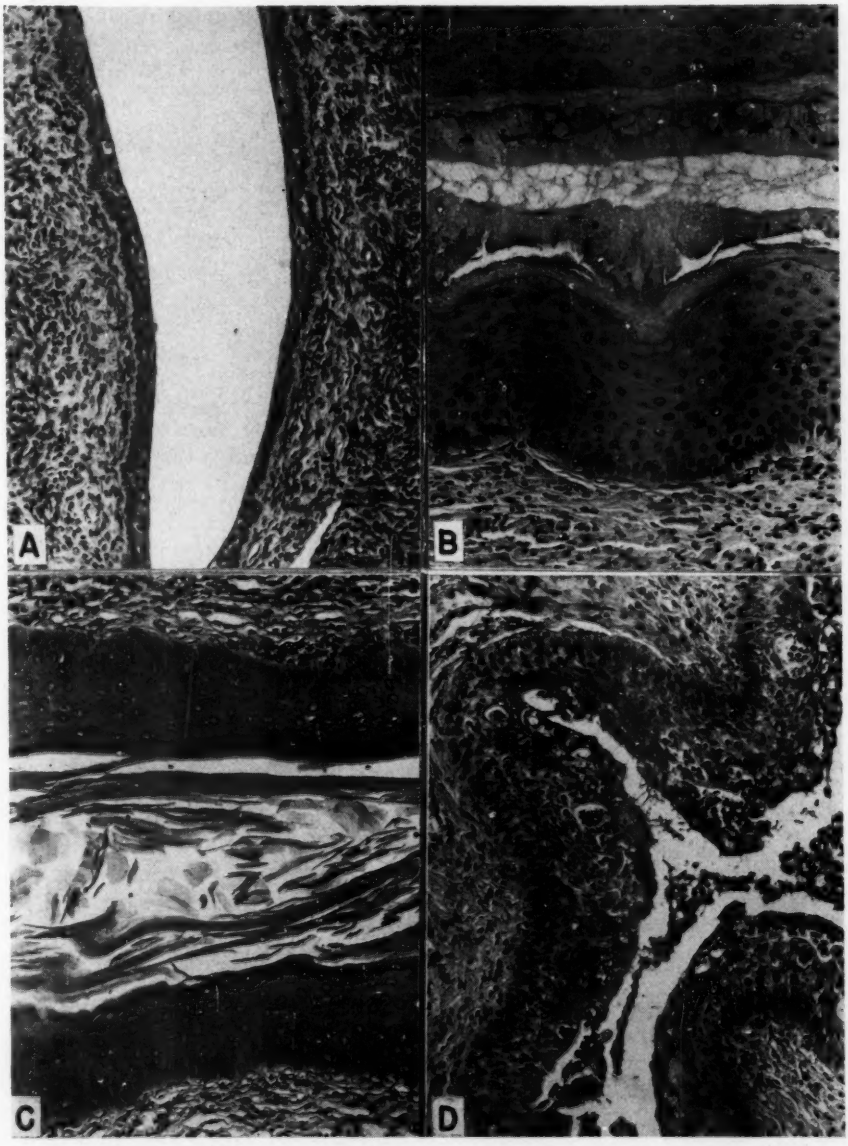


Fig. 1. Cyclical changes in the vaginal mucosa of control mice. (A) Diestrus. (B) Proestrus. (C) Estrus. (D) Metestrus. (Hematoxylin and eosin. Orig. mag. X 400)

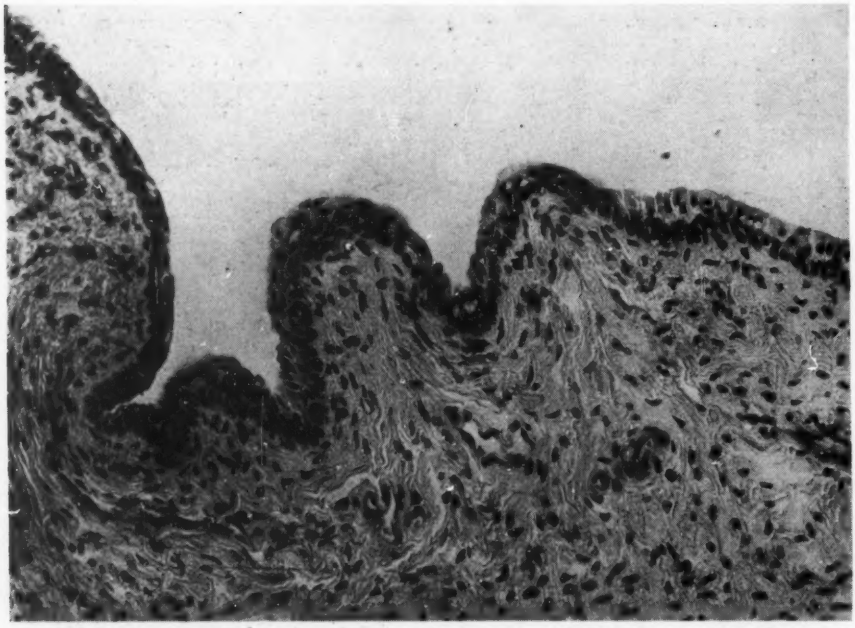


Fig. 2. Inactive vaginal mucosa (anestrus) typical of ovariectomized animals. (Hematoxylin and eosin. Orig. mag. X 360)

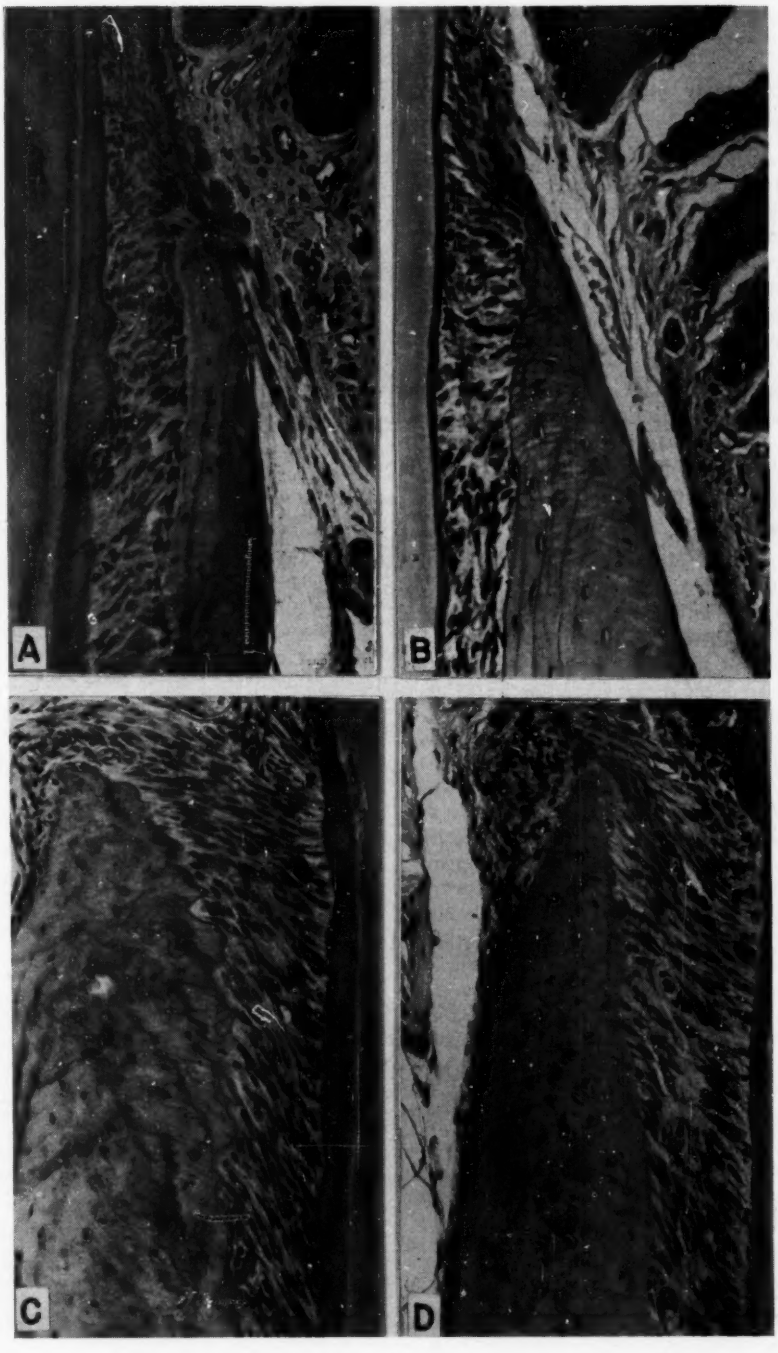
The gingiva in the ovariectomized animals presented no uniquely distinguishable microscopic features.

Changes in the periodontium were observed only in the animals ovariectomized at 4-6 weeks of age. The periodontium of the one year old experimental animals was unaltered. The earliest microscopic evidence of change in the periodontium occurred three months following operation. There was some variation in the severity of periodontal change in animals sacrificed at the same post-operative interval. However, in general, the changes became more pronounced as the post-operative interval increased.

DISCUSSION

Retardation in the normal alveolar bone formative activity adjacent to the periodontal membrane and diminished cellularity and fiber density in the periodontal membrane described following ovariectomy in an earlier report[1] were noted again in the present study. In essence the changes constituted a retardation in physiologic anabolic activity rather than the introduction of pathologic processes. The perio-

Fig. 3. (A) Control animal, 4-6 week group. Crest of lingual alveolar plate, mandibular molar, showing normal distribution of osteoid and osteoblasts lining bone adjacent to the periodontal membrane. (Hematoxylin and eosin. Orig. mag. X 400). (B) Ovariectomized animal, 6 months after operation. Crest of lingual alveolar plate, mandibular molar. The bone formative active normal for this area is absent. The periodontal membrane fibers are thinner, reduced in number and disorganized. The cellularity is diminished. Note the thin layer of cementum compared with control animal shown in 3(A). (Hematoxylin and eosin. Orig. mag. X 400). (C) Control animal, 4-6 week group. Crest of buccal alveolar plate. Note density of cells and fibers in the periodontal membrane, incremental lines in the alveolar bone and thickness of cementum. (Hematoxylin and eosin. Orig. mag. X 400). (D) Ovariectomized animal, 6 months after operation. Crest of buccal alveolar plate. Note reduced cellularity and filamentous appearance of collagen fibers in the periodontal membrane, sparsity of incremental lines in the alveolar bone and thin cementum as compared with control animal shown in 3(C). (Hematoxylin and eosin. Orig. mag. X 400)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dontal changes occurred only in the young adult animals, ovariectomized at 4-6 weeks of age. No periodontal changes followed ovariectomy performed in one year old animals.

Additional aspects of the effect of ovariectomy were explored in the present study. The post-ovariectomy time required for changes to occur in the periodontium was demonstrated to be three months. The changes in the periodontium became more pronounced as the post-operative interval increased. This appeared to be the result of a cumulative effect rather than in increased severity of the impact of ovariectomy upon the periodontal tissues in the latter days of the experimental period. The basis for such a hypothesis was most apparent in the cementum.

Retardation in the apposition of new cementum was noted after three months, but at this time, the thickness of the cementum in the control and experimental animals was essentially the same. At the six month interval the result of this retardation in cementum formation was evident. The cementum of the ovariectomized animals had attained a thickness which was only half that in the controls.

Also explored in this study was the possibility of a stimulation of cellular activity in the adrenals in an effort to compensate for estrogenic insufficiency from ovariectomy. Such adrenal activity has been reported in the rat,[3] but it has not been established that there was an associated increase in estrogen secretion. In ovariectomized mice, the findings in the adrenals vary with the strain.[4] Ordinarily, ovariectomy in the mouse leads to no detectable morphologic or functional changes in the adrenals.[5, 6, 7] In cancer-susceptible strains[8, 9, 10] ovariectomy may be followed by adrenal cortical hyperplasia and/or cortical carcinoma, with recurrence of the estrus cycle and development of the uterus and mammary glands.

In the R.A.P. strain used in this study there was no microscopic evidence of compensatory cellular activity in the adrenals following ovariectomy. In the vagina there was no recurrence of cyclical changes in the epithelium. In the periodontium, there was no adaptive reparative activity sufficient to reverse the effects of ovariectomy. In so far as could be determined by microscopic examination there was no evidence of an effective source of estrogen secretion in the ovariectomized animals.

SUMMARY

Two hundred and sixty virgin female albino mice, R.A.P. strain, in age groups of 4-6 weeks and one year were studied in the second in a series of investigations dealing with the effects of ovariectomy upon the tissues of the periodontium. Periodontal changes occurred only in the animals ovariectomized at 4-6 weeks of age and consisted of a retardation in the formation of alveolar bone and cementum and reduction in fiber density and cellularity of the periodontal membrane. The earliest microscopic changes occurred three months following ovariectomy and became more pronounced as the post-operative interval increased. There was no evidence of compensatory cellular activity in the adrenal glands following ovariectomy.

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Wire Ligature: An Undesirable Form of Temporary Splinting

by BERNARD B. SATUREN, D.D.S., *Philadelphia, Pa.*

MODERN periodontal literature is replete with recommendations for the use of wire ligature in the temporary splinting of mobile teeth: "The most satisfactory temporary splinting for anterior teeth is by the use of wire."[1] Wire ligature is "the most common form of temporary splint."[2] "Wire ligatures are employed when a more lasting temporary splint is desired."[3] "The anatomy of the anterior teeth is conducive to splinting by wire ligation,"[4] and . . . "the use of stainless steel wire ligature for temporary splinting of teeth for stabilization is an extremely beneficial aid in periodontal therapeutics."[5] And again, "Stainless steel wire ligation is indicated for the stabilization of teeth when such fixation is needed for a short to moderate period of time."[6] As a temporary form of splinting it may be used, according to our texts, for as long as two months;[2] or, as a "more lasting temporary splint,"[3] as long as several years[1] and even indefinitely.[7]

The implication gained after a study of texts is that one should make use of this technique because it is readily available, quickly manipulated, inexpensive, and efficient in maintaining stability of anterior teeth over short or longer periods of time. Very little is mentioned about the contraindications to the use of wire ligature. Wire is, of course, unsuitable for posterior teeth; its esthetics when used on upper teeth is undesirable; the problem of maintaining hygiene is mentioned; and the need for further adjustment is indicated. If subsequent adjustment due to loosening is necessary, it becomes evident that wire is *not* a good stabilizer of teeth. The confusion is heightened when one reads, "In order for the ligation to be effective it must be under tension."[8] Tension represents an active principle of movement and is obviously a disadvantage where immobilization —stabilization, lack of movement—is being sought.

The purpose of this paper is to prove conclusively that wire ligature is *undesirable* and should *not* be used for ligating teeth. It will purport to show that teeth while "stabilized" with wire ligature in accordance with accepted techniques of wiring will be extruded, intruded, will twist axially, will be malposed lingually or labially, will tip and buckle.

In considering the upper and lower anterior teeth from their facial and proximal aspects, it will be noted that their crowns

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Grateful acknowledgement is made to Mr. Alexander Mucha, Department of Visual Education, Temple University School of Dentistry for his help in the preparation of the diagrams.

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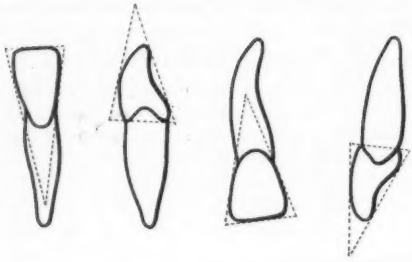

Fig. 1. Illustrating general cone-shapes of the crowns of anterior teeth.

present in general a cone-shaped appearance (Fig. 1). The cone appearance from the facial aspect has its apex pointing toward the apex of the tooth; the cone appearance from the proximal aspect has its apex pointing toward the incisal. Note that with each successive tightening a wire will slide along the converging walls toward the apex (Fig. 2). This is an illustration of the principle of component forces in physics. The ligature would remain at a given point governed by the cone-shape factor from the labial aspect being balanced by the cone-shape factor of the proximal aspect for each tooth individually. This "balance" would be true only if the convergence of the cone shapes of the two aspects were mathematically identical quantitatively. If several cones are placed adjacent one to the other and ligated together, it might be considered that the wire would be maintained in its position by the fact that it is part of the wire surrounding adjacent cones. Actually the cones will protrude out of the tightening

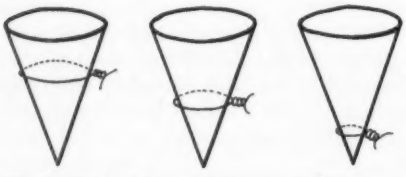

Fig. 2. Illustrating successive tightening of wire loop about cone with simultaneous slipping of wire loop in direction of apex of cone (based on action of components of force as a physical principle).

loops depending on their angles of convergence, the overall height of the wire being maintained (Fig. 3). As teeth are generally ligated by wire, note the possibility of their extrusion from the wire loops when viewed from the facial as well as their possible intrusion as viewed from the proximal aspects of each tooth contained within the loops of the ligated group of teeth (Fig. 4). Whether the result will be extrusion or intrusion depends upon two factors: first, the degree of convergence of the cones of the teeth; and second, the comparative degree of tissue support: a tooth with greater periodontal support will not be moved as readily as a tooth with lesser periodontal support.

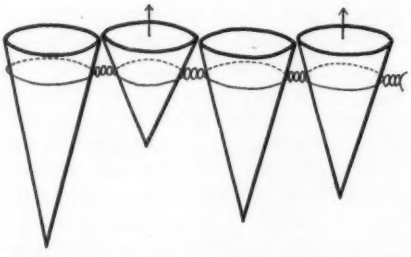

Fig. 3. Cone protrudes through wire loop on tightening, depending on angle of convergence.

Recognizing that the use of wire ligature involves active tooth movement, note now the proximal relationships of ligated teeth. All are aware of the convexity of the proximal surfaces of anterior teeth and that these teeth have a point proximal contact relationship. In the adjustment—tightening—of a wire about a group of teeth one must acknowledge a twisting (rolling) of one tooth against its adjacent tooth because of this convexity and point contact. This twisting would be noted especially where the arch of the teeth is not regular to begin with. All imperfections in the regularity of the arch curve would become exaggerated with the tightening of the ligature wire. With each succeeding future adjustment in making the ligature tight about the teeth, the irregularity would be more pronounced.

How does this twisting, this axial movement of the teeth under ligature adjustment, develop? In placing the main arch loop about the teeth, the wire is either pinched or bent to conform to the interproximal indentations or the smaller interdental wires that are placed will create these indentations. With the tightening of the interdental wires, the main arch wire presses against the facial and lingual aspects of the teeth. The point of contact of the proximating teeth acts as a fulcrum, and the teeth rock or twist as the interdental wires are adjusted tightly—that is, the teeth turn axially and individual ones may move lingually or labially. As the teeth turn or move out of the line of the

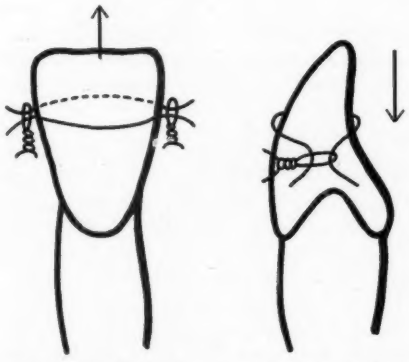

Fig. 4. Tendency for extrusion of tooth from facial aspect and for intrusion from proximal aspect.

curve, the arch of the curve becomes smaller, creating a space at the distal aspect of the ligated group of teeth on both the right and left sides, and the contained teeth assume a buckled, crowded arrangement. As the wire ligature is worn by the patient, it is noted that further adjustment is needed, not because the wire stretches but because the teeth are moved together or buckled. It can be assumed that the wire does not stretch because in adjusting ligature wires about teeth breaking of the wire is a common experience. The wire can be twisted only so much, after which it breaks. In many instances patients have returned with broken interdental wires which have had to be replaced—indicating that the added pressure of mastication was sufficient to cause breakage, not stretching. If there is a need for further adjustment of ligature wires, it indicates a movement of teeth closer together and not a stretching of the wire itself.

While fixation of mobile teeth is an important adjunct in the treatment of periodontal disease and helps to permit normal healing to take place, it is the purpose of this paper to show that wire ligature is not the proper means of achieving this end. Even though in the past it was deemed advisable to use this form of stabilization, it can now be concluded that wire alone should not be used for ligating mobile teeth. The use of wire induces active forces on the ligated teeth, causing them to assume new positions that result from extrusion, intrusion, rotation, labial and lingual malposition, and buckling. Today, with modern materials and techniques, teeth may be otherwise stabilized so that they are really fixed in position.

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Prophylactic Periodontics*

by CLIFTON O. DUMMETT, D.D.S.,** *Tuskegee, Alabama*

ANY discussion of basic considerations in the field of periodontics must begin with "prevention", which is defined as the act of keeping from happening. In medicine and dentistry, prevention implies the guarding against disease. A preventive then is an agent which wards off disease. A synonym for "prevention" is the term "prophylaxis". Through common and familiar usage the term has lost a portion of its pristine purity, and its usage has become somewhat restrained.

It is this subtle taboo, however, which has made the term deceptively interesting, has stimulated an even greater awareness of its connotations and its significance, and introduces this initial discussion of "Prophylactic Periodontics".

Like faith, prophylactic periodontics can move the dental profession out of its state of socio-professional tranquillity.

Like hope, prophylactic periodontics is eternally within the scope of all branches of dentistry.

Like charity, prophylactic periodontics begins at home with the dentist himself.

Ideally, the concept of prophylactic periodontics embodies the philosophy of eradicating eventually the current need for periodontists. Realistically, the practice of prophylactic periodontics is bound to utilize accepted measures and procedures in mitigating the ravages of those periodontal diseases which cripple the entire dental apparatus and drastically reduce masticatory efficiency.

*From a presentation to the Odonto-Chirurgical Society, Philadelphia, Penn., on Annual Clinic Day, April 29, 1959.

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ORAL PHYSIOPROPHYLAXIS

The treatment of oral disease by physical or non-medical means is referred to as oral physiotherapy. The underlying impression appears to be centered around "Therapeutics",—implying treatment after disease has occurred. This is rather different from the concept of utilizing non-medical or physical measures to prevent the occurrence of those oral diseases which are likely to exist in the absence of functional stimulation. Because accurate terminology is essential to adequate comprehension, the terms "Oral Physioprophylaxis" are introduced to include in this concept, those physical resources which should be prescribed by the dentist in order to prevent oral disease,—essentially periodontal disease.

The oldest and most firmly established physical implement for oral cleansing is the toothbrush. Basically, this agent consists of a small brush with a long handle. With the passage of time, there have been much experimentation with design, an enthusiastic promotion of universal distribution and an incessant exploitation of the public's gullibility. The large number and variety of toothbrushes on the market today are confusing to both dentist and patient. This confusion is heightened by the spectacular, but nevertheless unsubstantiated claims of resolute manufacturers who use the potent media of television, radio and motion pictures to advertise their products.

It would appear, therefore, that there is a distinct need for the dentist to evaluate scientifically the available information about toothbrushes, so that he would be in the best position to advise his patients.

It is the author's policy to recommend that patients procure three new toothbrushes every three months. It is not unreasonable to recommend a total of 12 toothbrushes per year for an employed in-

dividual, of middle class status specifically interested in his oral health as an indispensable unit of his general systemic well-being. Surely it would not be prohibitive to insist that there be a matching of the enthusiasm displayed over what are usually regarded as standard bourgeois perquisites with an interest in procedures associated with health and longevity.

The toothbrush bristles should be arranged with 6 tufts in length and 2 in width, preferably of natural texture, equal height and hard consistency. The handle should be straight and shaped for easy grip. Adequate bristle spacing facilitates ready cleansing. These requirements satisfy the criteria of utility, simplicity and effectiveness.

Man's insistence upon labor simplification has prompted the continual search for easier ways to accomplish his chores, so that over the years, there have been devised various ingenious mechanical and electrical contrivances,—all with the specific intent of reducing the physical energy utilized in brushing the teeth. This author feels that there are limitations to simplification, and furthermore, in the procurement of oral cleanliness, the use of contrivances per se does not automatically justify the supplantation of a desirable amount of physical exercise.

There are two special effects which toothbrushing is designed to promote. These are cleansing and massage. Were these functions more universally recognized, it is possible that there could be produced a closing of the wide gap which exists between the trite prating about brushing, and the actual use of a toothbrush,—at least 3 times per day every day.

Prevention demands that patients be informed of the fact that the process of cleansing requires all stagnant food material and other debris to be removed any time there is an accumulation, and accumulations occur whenever any food is taken into the mouth. The admonition that one should brush after meals, would appear, therefore, to be insufficient. It is preferable to modify this rule and advise that brushing should be accomplished immediately after any variety of dietary intake.

Impracticability is the word most often used to explain reasons for infrequent toothbrushing. It is true that the above program is somewhat time-consuming, and in the case of individuals who have the habit of constantly desiring some form of food material in the mouth, the frequency of cleansing activities would not be easily managed.

A precipitous diminution in such a habitual intake of food would automatically necessitate a decrease in the need for the associated frequency in cleansing. This alternative is undoubtedly the more practicable procedure, and from innumerable viewpoints, the more advisable.

Gingival massage is the methodical application of a stroking or kneading pressure to the periodontium, for the purpose of stimulating the blood circulation to these tissues. The activation of the local blood supply materially assists the process of nutrition and elimination of waste products. The ability of the local circulation to build and maintain a condition of health is an important factor in oral physioprophylaxis, and it has been recognized by periodontists as vital in promoting the clinical appearance of health. Emphasis is placed upon "clinical" appearance, because of the recognized division of opinion regarding the pros and cons of massage. The consensus of opinion of those who do not support the theory of the beneficial effects of massage may be summarized by the following statement of Lyons: "It is yet to be shown, by acceptable scientific methods of study, that rubbing, scrubbing, stroking, squeezing, vibrating or compressing and releasing the gingivae by brush, rubber cone or wooden stick contribute either to the rate or extent of healing. On the other hand, it can be shown that spectacular results are obtainable without these adjunctive measures. The contention that periodontal healing cannot be satisfactorily

induced without so-called massage is contrary to the facts".

The greater number of avowed proponents of massage can be attributed to the fact that clinical studies have indicated that massage of the gingiva with a toothbrush increases keratinization of the surface epithelium, thereby providing greater protection against trauma. Also, massage provides a reduction in gingival inflammation.

Equally confusing to the conscientious consumer of oral health instruction are the numerous methods of toothbrushing, and once again it is essential to insist upon simplicity in any instructions given to patients. Recognizing the plethora of toothbrushing technics, all of which possess many aspects to support their being recommended, it would appear that the most serviceable and practical method is one which insists upon brushing the upper teeth downwards, starting way up on the gingiva, and brushing the lower teeth upwards, starting way down on the gingiva.

Of even greater importance than these simple instructions is the singular factor of demonstrating the technic upon the patient himself. This procedure gives the opportunity for the dentist to check on whether the patient has purchased the correct size, type, form and number of toothbrushes. The author has found frequently that patients' interpretations of toothbrushing pressure are vastly different from the pressures they experienced when he personally brushed their gingivae and teeth.

In physioprophylaxis, the topic of dentifrices and mouthwashes can be summarily dismissed with the suggestion that persons should use the American Dental Association approved products for which there may be a personal preference. Emphasis should be placed upon the fact that these agents are cleansing and polishing aids, which do exert some mechanical rinsing action, but they should not be regarded as substitutes for the toothbrush itself. Recognizing that calculus remains one of the most potent forces to be counteracted in the prevention of periodontal disease, it is reassuring to know that extensive research is in progress to produce a dentifrice which will act as a "calculytic" agent. One of these agents to which much attention has recently been directed is Extar. Although many reports of its use have been encouraging, Thomas indicates that this enzyme-containing dentifrice does not prevent materially the continued deposition of calculus in inaccessible areas. Some preventive action is discerned in those areas which can be easily reached and mechanically cleansed with the toothbrush.

Of even greater physioprophylactic significance would be the discovery of an "anti-calculogenic" agent which would prevent materially the formation of calculus in the first place. Such anti-calculogenics will probably depend upon more authenticated information concerning the etiology of calculogenesis.

Although the toothbrush is an excellent instrument for massaging those tissue surfaces that are adjacent to the lips, cheeks and tongue, there are many occasions in which it is difficult to massage the interdental tissues as effectively with the brush. Under conditions of physiologic normalcy, these tissues usually fill the interdental spaces up to the contact point. Therefore, interdental massage would not normally be employed, and should not be forced upon the tissues. In those cases in which there has been a functional recession of the tissues, the interdental stimulator may be utilized, gently but effectively, both to cleanse and to stimulate. Its use should be recommended and demonstrated by the dentist.

The small pyramidal rubber tip at one end of a large number of the toothbrushes now being manufactured, is probably the most effective of interdental tissue massaging agents.

The effectiveness of the dentist in convincing persons of the desirablity of

physioprophylactic procedures is intimately associated with the oral health practices of the dentist himself. The maxim,—"Example is better than precept"—is nowhere any more eloquently punctuated than in the dynamic situation in which a patient conference on the elimination of halitosis, for example, is conducted by a dentist, himself "standing in the need of aid". The picture of dentists, themselves requiring extensive dental care is not an infrequent occurrence. Without belaboring the point, it should be emphasized that the most convincing argument for instituting procedures to maintain a condition of oral health lies in an equally convincing demonstration of actual results of those procedures in the dentist himself.

RELATION TO OTHER BRANCHES OF DENTISTRY

The integrity of the periodontal tissues is intimately associated with the health of the other tissues comprising the dental apparatus. This integrated relationship demands that there be maintained the delicate balance between procedures instituted for the correction of oral disease and deformity on the one hand, and the status of the periodontium on the other. The implication of a sustained high quality of dental therapeutics, is therefore loud and clear.

Dentists are aware of many of the abuses to which the periodontium is frequently subjected. These range from those auric anterior odontological decorations with pernicious gingival margins, through other violations which result from accidents or altercations, to those misuses that are self-inflicted. The teeth are the oral units most often implicated in these abuses. The use of the teeth to suspend the human body in acrobatics and their use to bite and chew excessively hard objects are examples of self-inflicted abuses which can and do produce deleterious effects on the supporting tissues. From a prophylactic viewpoint, it is the very acme of simplicity to urge patients to avoid trauma by abstaining from these activities which are capable of yielding such devastating consequences. Similar advice should be directed against utilizing excessively hot or excessively cold foods and other substances capable of producing injury to the pulpal tissues. This type of injury may be complemented by alterations in the surface epithelium of the mucosa.

In addition to the dentist-induced irritations produced from the overhangs and margins of restorations, and the results of injurious habits, there are frequently listed among the many local etiological agents of periodontal disease, such factors as malalignment, malocclusion, food impactions, loss of teeth without replacements, mouth breathing, functional insufficiency and traumatic occlusion. The treatment of these abnormalities comes within the purview of the orthodontist, the operative dentist, the prosthodontist, the restorative dentist, the pedodontist, oral surgeon and the general practitioner of dentistry. It is obvious then why the prophylaxis of periodontal disease falls within the scope of every branch of dentistry.

DIET AND NUTRITION

A diet is a prescribed course of food, and the methods by which the human body utilizes this food to promote the processes of growth and development and to repair damaged tissues are known as nutrition. Nutrition involves the ingestion, utilization and metabolism of carbohydrates, fats, proteins, vitamins and minerals. When the nutritional state is normal, there is no impairment in tissue function and structure. In nutritional deficiencies, however, there is an inadequacy in the nutritional state of the tissues,—the periodontium included. Such a deficiency status may result from an insufficient amount of food or from some disturbance in the process of ingestion, utilization and metabolism.

Prophylactic periodontics would require that there be the correct quality and quantity of food eaten, in addition to the assurance of an absence of any disease conditions

which may adversely affect the nutritional process. This latter requirement is of course the more difficult, and is thoroughly dependent upon a close and cooperative relationship, not only with the physician, but also with every other member of the health team.

Insofar as the quality of ingested food is concerned, it is the consensus of opinion that there are beneficial effects from the stimulation afforded by the chewing of those types of food which exert a detergent action on the periodontium. The studies of Pelzer, King and Glover, and Haydak and associates carried out both on experimental animals and humans, are among many which have provided the evidence to conclude that soft diets are injurious to the periodontal tissues, since they afford neither a functional stimulation nor a natural dispersion of accumulated food remnants around the gingival margins. From the physioprophylactic viewpoint, therefore, vigorous chewing as required by detergent foods is a necessary and effective expedient.

Basic to the considerations discussed so far is the realization that in prophylactic periodontics, there are many of the human factors which contribute to the acknowledgment that dentistry is a scientific profession with social aspects. There is the need to appreciate each patient as a distinct personality, as a member of a family, living in a locality which may have an effect upon his general and dental health.

One of these human factors is the question of human lethargy. There is a certain amount of general indifference which the dentist must conquer, first within himself, then on the part of the patient. The aforementioned factors of the impracticability of frequent toothbrushing, dietary habits and the presence of faulty dentistry,—are examples of this indifference.

PUBLIC HEALTH ASPECTS

So far the discussion has been confined to the individual approach in prophylactic periodontics, and emphasis has been placed upon what should be told each patient to help him or her to prevent the onset of periodontal disease. If the ratio of dentist to patient and the distribution of dentists were more equitable, this individual approach would of course be unique. In the absence of such idealism, however, it becomes necessary to promote some other approach whereby a larger number of persons could be reached and advised about these matters. This is the public health approach.

In considering the public health aspects of prophylactic periodontics it is necessary to admit first that periodontal disease is a public health problem.

Chronicity, wide prevalence and high cost of care, these are among the factors which support the contention that certain phases of periodontics constitute a public health problem.

As such then, it is necessary to utilize public health methods in its solution. There can be no mass application of technical procedures in periodontal therapeusis. The unit of operation is a single person necessitating individuality of treatment by a particular clinician. Many of the procedures and recommendations that have been discussed in prophylaxis lend themselves to the public health educational approach. Among these are dietary regimen, oral hygiene, the effects of irritants, home care, health demonstrations and massage. In a very real sense, their institution can be developed as an educational responsibility requiring community-wide health educational effort. Furthermore, there can be utilized the same auditory and visual educational media which manufacturers use in promoting toothpastes, gargles, mouthwashes and all of the other products which are recommended for the attainment of an oral "Shangri-la".

The necessity for periodic dental examinations must be explained to the public. This is a primary function of the health educator, but this fact does not in any

way lessen the responsibility which dentists and other members of the health team have in this regard. Physicians, nurses, hygienists and medical social workers should be educated about the elementary facts in care and prevention. They must be especially mindful of the fact that programs of prevention must be based upon a thorough knowledge of causative factors and epidemiological characteristics. Inasmuch as there is still the great need for research into these areas, it is therefore necessary to caution health educators in the dispensation of knowledge. The public must be informed about what is fact and what is fiction.

One final consideration in this matter of "prophylaxis" is in need of delineation. The chances are that if these recommendations for periodontal disease prophylaxis are diligently, faithfully and continuingly followed, there would not be the high incidence of periodontal diseases that there exists. Certainly there would be a precipitous decrease in those forms of periodontal disease which have their origins in local irritation. Because there are still unknown areas about etiology, however, there is every reason to expect that some forms of periodontal disease will continue to occur. There is need then to think about prevention insofar as the progression of incipient periodontal disease is concerned. The prevention of further alveolar destruction, of increased mobility, of purulent discharge and of crevicular epithelial ulceration thus becomes a matter of periodontal therapy. Next step in the hierarchy of "prophylaxis" would be the prevention of tooth loss, the ultimate goal of dentistry in general and periodontology in particular. Insofar as periodontics is concerned, this becomes a function of a genuine alliance between periodontal therapeutics and the technical procedures of other branches of dentistry.

The completion of the prophylactic cycle is afforded by the full realization of the importance of intercepting any noxious consequences of periodontal disease which may disturb the patient's general systemic well-being.

SUMMARY

1. There has been suggested the term "Oral Physioprophylaxis" to include those physical resources which the dentist should prescribe to prevent oral disease.

2. There is a definite need for the dentist to evaluate the available information about toothbrushes so that he would be in the best position to advise his patients.

3. Prevention requires patients to be informed that cleansing requires the removal of all stagnant food material and other debris any time there are accumulations, and these occur after each period that any food is taken into the mouth.

4. Clinical studies have indicated that massage of the gingiva with a toothbrush increases keratinization of the surface epithelium, thereby providing greater protection against trauma.

5. Of great importance is the factor of demonstrating a toothbrushing technic on the patient himself.

6. Dentifrices and mouthwashes exert some mechanical rinsing action, but should not be regarded as substitutes for the toothbrush.

7. The most convincing argument for instituting oral health procedures lies in an equally convincing demonstration of actual results of those procedures in the dentist himself.

8. Faulty dentistry can no longer be excused, condoned or protected, either by the responsible dentist or his colleagues.

9. Because of its chronicity, wide prevalence and high cost of care, periodontal disease is a public health problem.

10. There is a prophylactic periodontics cycle which includes (a) preventing the initiation of periodontal disease (b) preventing the progression of periodontal disease (c) preventing tooth loss (d) pre-

venting noxious systemic consequences of periodontal disease.

CONCLUDING STATEMENT

In conclusion, the concepts discussed are all part of the basic philosophy of modern dentistry. Qualified dentists have a duty to espouse the profession's progress. As dentistry embarks upon its second century of existence, it is paramount that dentists renew resolutions to think of it with loyalty, to extend its virtues and to continue to act always as guardians of its hard-won reputation.

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Systemic Lupus Erythematosus
A Seven Year Case Report*

by MARVIN M. SUGARMAN, D.D.S.** AND H. B. STILLERMAN, M.D.***, *Atlanta, Georgia*

SYSTEMIC Lupus Erythematosus is a disease of interest to the dentist as well as the physician because the lesions may initially appear in the oral cavity, and the oral lesions afford a vexing problem of therapy. Since this is a highly fatal disease, without therapy prompt recognition of this illness would benefit the patient tremendously, enabling him to receive early treatment.

During the past decade Systemic Lupus Erythematosus, a collagen disease, has been diagnosed with increasing frequency. Kaposi[1] originally described the disease in 1872. As early as 1905, Osler[2] reported the various manifestations as follows: "By Exudative Erythema is understood as a disease of unknown etiology with polymorphic skin lesions, hyperemia, edema, and hemorrhage — arthritis occasionally, and variable number of visceral manifestations of which the most important are gastro-intestinal crisis, endocarditis, pericarditis, acute nephritis and hemorrhage from the mucosal surface. Recurrence is a special feature of this disease and attacks may come on month after month or even throughout a long period of years. The attacks may not be characterized by skin manifestations. The visceral symptoms may be present and to the outward view the patient may have no indications whatever of erythema exudativum." Diagnosis of this disease was facilitated in 1948 when Hargraves [3-4] described the L E cell. Rein, Kostant[5], and Moore[6] found the occurrence of biologically false positive serologic tests for syphilis in patients with Systemic Lupus Erythematosus. In approximately 14%[7] of cases, oral lesions occur. These oral lesions[8] initially show an area of bright redness, the surface of which is soon torn by excoriation and later ulcerates. After the red lesion has been present for some time, numerous white lines appear. The larger patches show deep central fissures or ulcers. The margins have whitish satellite areas ranging from pinpoint to pinhead in size. In older lesions the red border becomes less elevated and is transformed into a white or bluish-white peripheral zone of thickened epithelium. The lesions enlarge by healing in the middle and breaking down at the periphery. The lesions are found most often in the mucobuccal folds and are usually bilateral. After healing there remains a scar tissue which is very easily traumatized.

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CASE HISTORY

The patient (a white female) was first seen in March, 1951. At that time she was 28 years old. Her chief complaints were chills, fever and night sweats. There was a past history of rheumatic fever, thyroidectomy, and numerous genito-urinary tract infections. Physical examination revealed a fever of 104° F, generalized glandular enlargement, an injected throat, pain over the frontal sinuses, and a systolic murmur in the aortic area. There were no petechiae or rash present. The spleen and the liver were not palpable. She did not respond to oxy-tetracycline at home, and was hospitalized with a tentative diagnosis of bacterial endocarditis. Hemotological studies revealed a leukopenia of 3,600 with a normal differential, and 11 gm. of hemoglobin. Blood cultures and cold agglutination tests were all negative. Electro-cardiogram and roentgen examinations of the chest were normal. She responded to intra-venous chlor-tetracycline. She was discharged with the diagnosis: fever of unknown origin. In two or three weeks another relapse occurred with chills, fever, and night sweats. There were questionable petechiae in the conjunc-

Fig. 1

Fig. 2

tivae. Again she was hospitalized for study. Eleven blood cultures were negative. A urine culture grew out an E. Coli and an Enterococcus. These organisms were sensitive to oxy-tetracycline and she was treated with this drug at home and at the hospital. Her temperature become normal. After five days it was elevated again. Cystoscopic examination and retrograde pyelograms were normal. Early in April, her temperature again rose to about 101° F each day. All of the blood studies and agglutination series were normal. There was generalized glandular enlargement. A lesion on her arm was suggestive of erythema marginatum. Her husband stated that the degree of fever depended on the amount of activity. Oxy-tetracycline now had no effect. This time the possibility of collagen disease was considered. She was again hospitalized in June, 1951. At this admission, a physical examination showed a papular lesion on the base of the nose, over both shoulders, on the arm, and several oral lesions. (Figs. 1, 2). These oral lesions demonstrated an erythematosus base with the whitish satellite areas. A deep red color on the tongue was apparent. No petechiae were seen. There was generalized glandular enlargement still present. The laboratory studies still showed a leukopenia, negative urinalysis, negative tuberculin test, and negative blood cultures. Lymph node, bone marrow and muscle biopsies were performed. These confirmed the diagnosis of Systemic Lupus Erythematosus. In addition, there were L E cells in the peripheral blood.

Fig. 3

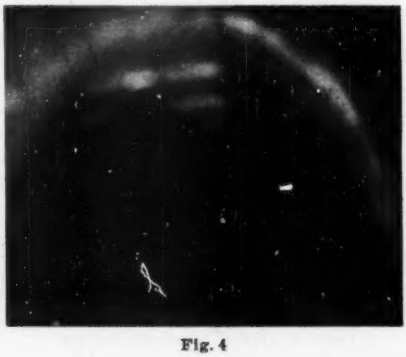

Fig. 4

Fig. 5

The patient was treated with ACTH and then was changed to Cortone. In October, 1951, steroid therapy was discontinued. She progressed well for about three weeks. The oral lesions healed in the center and broke down at the border, (Fig. 3). At that time she exposed herself to the sun. The rash and fever reappeared. The oral ulcerations, (Fig. 4) were large and painful. Again she was brought under control and healing of the lesions occurred with the use of steroids. Since then she has been on steroid therapy continuously. The lesions almost disappeared; however, the disease remained active. The deep red color remained around the lips, (Fig. 5).

Fig. 7

Lesions both oral and dermal appeared intermittently. There has never been any renal involvement. The erythrocyte sedimentation rate has been elevated, and she has remained on a low salt, high protein diet. The hemoglobin has remained within the normal range. The L E cells have always been demonstrated with ease. In April, 1953, the patient again exposed herself to the sun for a period of 45 minutes. A marked rash and fever developed with the fever as high as 105°F. The facial and oral lesions both showed marked ulcer formations. (Figs. 6, 7). She was hospitalized in a moribund state. Treatment con-

Fig. 6

Fig. 8

Fig. 9

sisted of intra-venous infusions of ACTH and antibiotics. The oral lesions were so painful that eating was almost impossible and anesthetic lozenges were used. Her skin and oral lesions, which had become secondarily infected with staphylococci, responded to treatment with erythromycin ointment. (Fig. 8). An oral monilial infection occurred and healed uneventfully. In June, 1953, she was discharged from the hospital, and the infection was completely controlled. She was being maintained by either 100 mgs. of cortone or 40 to 80 units of ACTH daily. Demoral was used for the relief of pain. Her fifth hospital admission was in April, 1954. At that time she had a normal complete blood count and sedimentation rate. L E cells were present in the peripheral blood. Roentgenographs and cardiograms were normal. The non-protein nitrogen was slightly elevated, being 50 mg. per cent. A remission and healing again took place. Her mouth and hands had a few lesions. She looked well, had a good appetite and a normal temperature. A mild generalized glandular enlargement was still present. The spleen was not felt. Exposure to the sun was avoided completely.

The sixth hospital admission was in March, 1955, for routine evaluation. The peripheral L E cell was absent for the first time. Her non-protein nitrogen was 42 mg., WBC 7,400 with a normal differential, hemoglobin was normal, erythrocyte sedimentation rate 12 mm. per hour. The cardiogram was normal and the urinalysis was negative. In April, 1956, the patient was again admitted to the hospital with severe herpes zoster of the left ophthalmic nerve. (Fig. 9). She was treated with intravenous ACTH infusions and penicillin. Recovery was uneventful and she was dismissed from the hospital with instructions to continue her cortone therapy. Meticortin caused increasing tension and was discontinued. In September, 1957, the patient was admitted for neuro-surgical consultation. The pain from her herpes zoster was excruciating at times. At this time she had been controlled at thirty milligrams of Cortone a day. She was on a salt-free diet and was taking vitamins, sedatives, and potassium chloride. Laboratory studies revealed hemoglobin 13.6 gm., WBC 8,900 with a normal differential and had adequate platelets. The C-reactive protein tests showed a trace; thymol turbidity was 1.8 units; Erythrocyte sedimentation rate was normal. Roentgen examination revealed pulmonary emphysema and obliterative pleuritis of both bases with chronic changes. The heart was within normal limits. The cardiogram was normal. A phlebitis of the left foot and leg occurred. On September 24, 1957, the patient had a left middle cervical sympathectomy. The procedure was well tolerated and it relieved the headaches.

Fig. 10

Fig. 11

At present, the patient is maintained on 25 to 50 mgs. of Cortone per day. Additional medication includes potassium chloride, vitamins, and meprobamate. There are very few oral lesions; her palate, lips, fingers, and face remain relatively clear and she is able to carry out all the normal duties of a housewife. (Figs. 10, 11).

COMMENT

A patient with Systemic Lupus Erythematosus has been presented. The management of this case shows many of the difficulties and complications which may arise. Clinical manifestations included lesions of the oral cavity, face, conjunctivae, hands, head and joints. At no time has the disease been in complete remission.

Despite the smoldering process she has progressed extremely well. Her longevity is due to the fact that this illness has not attacked a vital organ, such as the kidney. The laboratory studies consistently revealed active disease as measured by the L E cell, sedimentation rate, and the C-reactive protein tests. As the history pointed out, exposure to sunlight will cause an exacerbation of the signs and symptoms of the acute disease.

Therapy in this disease has been revolutionized with the advent of the newer steroids. At first, Adrenal-Cortico-Tropic hormone was used, followed by various forms of oral steroids compounds. The patient has been easily maintained on the oral steroid compounds. The complications of steroid therapy must always be considered. Susceptibility to infection is increased and the steroids mask febrile reactions. The danger of adrenal cortical atrophy is always present. Despite these facts, steroids are the drug of choice for therapy in this illness. Another form of therapy advocated is the use of potent antimalarial drugs. The pain due to the ulcerated oral lesions was controlled by the use of anesthetic lozenges before meals. The oral lesions in this case always had exacerbations and remissions in direct proportion to the severity of the disease. Herpes Zoster Ophthalmia was purely coincidental. Since a patient with Systemic Lupus Erythematosus is markedly susceptible to infection, the prolonged use of antibiotics is frequently required. Untoward reactions may occur as demonstrated by this patient contracting oral monialiasis.

The steroid compounds have radically changed the prognosis of Systemic Lupus Erythematosus. There is no doubt that this patient was twice in the terminal state. ACTH and its allied compounds were responsible for her recovery. Although seven years have passed, it is still impossible to state the duration of life expectancy of this patient.

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The Dental Health of a Group of Handicapped Adolescent Children

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THE handicapped children in this study were selected on the basis of their ability to perform a considerable degree of self help in dressing, walking, eating, and personal hygiene. There were no epileptics nor mentally deficient in the group, although some were scholastically retarded on account of their inability to follow a routine schooling procedure because of their crippled condition or the duration of the illness which produced their condition. For example, in the post-polio group there were many cases of an interruption of several years in the normal education program.

This study is based on the findings in 122 adolescent children between the ages of 12 and 17. In this group there were 58 girls and 64 boys. No record was kept of nationality, race, or color. The children were from average homes, most of them in the low income bracket, and from small communities. Forty-five were inflicted with cerebral palsy, 52 were post-polio, and 25 had miscellaneous handicaps. These included Perthes disease, muscular dystrophy, spina bifida, scoliosis, roto scoliosis, Friedreich's ataxia, spastic paraplegia, hydrocephalic ataxia, Erb's palsy, bulbar palsy, multiple sclerosis, rheumatic fever, and others.

Lifetime dental health prospects are based to a considerable extent on foundations built during childhood and the adolescent period, following the same pattern that physical development and systemic disease beginnings have on the general stature of adult health. Growth changes speed up at this period of physical development. According to Krogman (8) the real important acceleration in growth from the standpoint of the dentist is the period from 10 to 15 years of age. The remaining deciduous teeth are now exfoliating and most of the permanent ones have erupted. The occlusion of the teeth is changing in accordance to the demands of function and stress. The gingival tissues which have gone through the stages of supporting the deciduous teeth and are now reforming in relation to the erupting permanent ones. Any abnormality of tooth position now requires compensation in the gingival fibers. For this reason when periodontal disease develops in the handicapped child, it progresses more rapidly with greater disability possibilities than in normal children.

Adolescence has an increased dental disease potential even in the normal child because of personal and parental neglect, but in the crippled child with a soft diet it is even greater. The gingival condition of this group under study indicates an extremely high percentage of beginnings of periodontal disease. The periodontal involvement was evaluated on a 0-1-2-3-4-basis as follows: 0 indicated good oral hygiene, evidence of adequate brushing, firm gums, normal color and stippling; 1 the beginning of color change with gingival thickening, a marginal inflammation, some calculus accumulation, interproximal redness, slight bleeding on brushing, slight pocket formation and some smoothing of stippling; 2 a definite rolling of the gums, bleeding of the gingival tissues on pressure, loss of stippling, alveolar crest involvement, debris on the teeth with wide spread calculus formation; 3 painful rolled gums which bleed on touch, pain, deep red color, flattening of interproximal papilla, some ulceration, loosening of teeth and position teeth changing or drifting; 4 mobile teeth, dark red gingiva which bleed easily, some ulceration, tissues painful, hypertrophic gums tissues covering a large part of the crowns of the teeth. The two cases in category 4 were under continuous treatment with dilantin but were not epileptic. The following chart summarizes this data. Chart One.

These figures indicate that only 18 or 14.8% were free of gingival disturbance,

while 63 or 50% have a definite gingivitis or periodontal disease which requires treatment and also indicates a lack of elementary oral hygiene procedures. Twenty-three of the children were in real trouble with advanced periodontal disease in category 2, 16 needed urgent and extensive treatment. Most important is the fact that on a total basis, 85.2% of these children had some degree of periodontal involvement. This data indicates a much higher percentage than is found in the normal average child, but there are many contributing factors in this group of children.

Diet undoubtedly plays a considerable part, for many of these children have been fed a soft diet as being the one which is easiest for the child to manage if the child is a self feeder, and for the parents if they have to feed the youngster. A diet which requires more chewing and use of the masticatory muscles can change this periodontal disease picture considerably. The studies of Newman, Lefkowitz and De Salvo (12) indicate this possibility. They found also that abnormalities of occlusion and habits, muscular spasms, sore teeth, etc., contribute. Ray and Santos (14) report that tongue thrusting which is a common habit in this group of children, especially those with cerebral palsy is a decided factor. Lite, Di Maio, and Burman (5) find that there is considerable gingival pathosis in the mouth breathers and those with open bite.

The adolescence period presents an increased caries potential not only because of the constantly changing oral conditions, but also because of a neglect pattern and probably a lack of health consciousness on the part of the teenager, at least in the earlier part of the period. These facts have been developed in different ways in other studies. Those of Muhler et al (11) are an example. In their control group the DMF index jumped from an average of 1.99 in the younger children to 4.93 in the adolescent group. Their studies also show that a moderate amount of preventive measures and dental health education can cut the caries process to a considerable degree, bringing the DMF index down to 1.17 in the children under ten and to 2.20 in the teenage group.

In this group of 45 cerebral palsy cases, 33 children had no evidence of previous dental treatment other than extractions to relieve pain or other conditions. There was evidence that much of this loss of teeth was actually due to difficulty in obtaining other adequate dental treatment in these cases, Lyons (7). These children had, at the time of examination, 104 decayed unfilled permanent teeth, 28 had been previously extracted, and 5 had periapical abscesses with gingival fistulas. This means a total of 137 involved teeth of the permanent dentition. Six children had had previous dental treatment with restoration of all carious teeth, and six others had no carious teeth.

In the post-polio group, 36 children had 64 carious permanent teeth, 18 teeth had been previously extracted, and there were 2 diseased molars, making a total of 84 involved permanent teeth. Sixteen of this group were caries free.

In the miscellaneous group the 25 chil-

CHART ONE

Cases	*Number of Children*	*0*	*1*	*2*	*3*	*4*
C.P.	45	3	22	13	7	1
P-P	52	13	25	5	3	1
Misc.	25	2	16	5	6	0
Totals	122	18	63	23	16	2

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CHART TWO

Cases	*Children*	*Carious Perm.*	*Prev. Extracted*	*Abscessed*	*Caries Free*
C.P.	45	104	28	5	6
P-P.	52	64	16	2	16
Misc.	25	73	13	2	9
Totals	122	241	59	9	31

dren had 73 decayed permanent teeth, 13 had been previously extracted, and there were two abscessed teeth. Nine of the group had no caries.

It is probable that many of the 241 carious teeth reported will be subsequently lost if the present inadequate program of care is continued, or a total of 308 lost permanent teeth before the age of 16. In some cases there will be other teeth involved with caries soon. Only 33% of the cerebral group, 47% of the post-polio group, and 38% of the miscellaneous exhibited any evidence of previous dental treatment, yet all these children were under the care of agencies presumed to be concerned with their health rehabilitation.

Teeth were considered to be in malocclusion or at least in functional imbalance if the anterior teeth were separated, very crowded, any tooth out of alignment or transposed, the bite open, etc. Thirty-five of the 45 in the cerebral palsy group or 77% were thus involved. Malocclusion is a very serious problem in this group at all ages, as previously reported the all age figure is 84% (6). 56% of the post-polio group and 45% of the miscellaneous group had some degree of malocclusion which contributed to the high incidence of periodontal involvement reported here.

SUMMARY

This rather limited study points up a number of pertinent facts regarding a special group of teenage children. Since this group were examined at a crippled children's summer camp it can be readily understood that these children belong to the self help classifications suggested by Wallace et al (13) and should be the best type for a rehabilitation program. Every day maintenance of dental health is paramount, but it must be on a long term continuation basis. For an example of this statement Krogman (9, 10) points out that there are about 11,000 children born every year with cerebral palsy, 1500 of these die in infancy, 3500 will remain in institutions throughout life, but of the 6000 that remain in society many can be trained to become self supporting useful members but their usefulness depends upon esthetic acceptance, personality, and the completeness of the rehabilitation program.

Complete and total dental maintenance of this whole group of children is very important if no other reason is considered than the fact that any diminution in esthetics adds a burden to the already psychologically impaired child. There are many possible methods of reducing the DMF index in these children. The fluoridation studies of Bushel and Smith (1), and of Brown et al (2), Muhler (11) indicate one possibility. The problem of obtaining adequate care for these children is complicated by the fact that few dental schools give adequate training to dental students in the care of handicapped children. Castaldi (3) reports that only a third of the schools have such a program. Social agencies instrusted with the care of these children must learn that adequate dental care does not mean emergency extractions but must include complete restoration of teeth and function as needed. The dental problems of this group are more acute and more destructive during the teenage than later in life.

A community problem also exists which

must be solved. Many of these children can only be cared for under a general anesthetic-hospital condition, yet few community hospitals have the equipment to permit dentists to restore teeth under such conditions, nor do they have the trained attending or visiting dental staff to make possible such a program.

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SEVERAL RESEARCH TEACHING FELLOWSHIPS AVAILABLE

Tufts University School of Medicine has recently announced that two research fellowships in oral pathology and periodontology are available for the school year beginning September 1960. Additional information and an application blank may be obtained from Dr. Irving Glickman at 136 Harrison Avenue, Boston 11, Mass.

The University of Pennsylvania and the University of California Schools of Dentistry are also offering fellowships for 1960-61. These fellowships are designed primarily for graduate dentists who are interested in a teaching career. Additional information may be obtained by writing the University of Pennsylvania School of Dentistry Committee on Fellowships and Traineeships, 4001 Spruce Street, Philadelphia, Pennsylvania, and Dr. Herman Becks, Chairman, Research Teacher-Training Program, University of California Medical Center, School of Dentistry, San Francisco 22, California.

Pain Control in Periodontal Surgery

by FRANK T. SCOTT, D.D.S., JACK K. WHITMAN, D.M.D., *Jacksonville, Florida*

MOST patients fear the pain anticipated in association with dental procedures. The successful control of emotional tension has, therefore, been found to be equally as important as the control of physical pain.

This report presents certain procedures which in the hands of the authors have proved highly successful in the management of apprehension, fear, and pain before, during, and after extensive periodontal surgery. This paper covers observations of cases encountered on our service over a period of years. Surgical procedures ranged from simple conservative gingivectomy to extensive advanced periodontal surgery. The diagnostic categories included osteoplasty, osteotomy including removal of tori, and vestibular surgery. One to four quadrants of the mouth were involved in the composite series.

PREMEDICATION

For minor cases of short duration premedication with an elixir of butabarbital sodium, 100-200 mg., administered orally thirty minutes prior to surgery, has proven very satisfactory. Patient acceptance has been gratifying and side-effects minimal. Patients receiving this medication in the dosage range mentioned have been able to go about their usual routine following surgery.

More complicated cases involving extensive surgical procedures require more potent and longer-acting sedative-analgesic medications. Patients in this category receive premedication approximately one hour prior to administration of the local anesthetic. A combination of four drugs which provide a unique synergistic effect is employed. These comprise:

Pentobarbital Sodium	50 mg.
Meperidine Hydrochloride	25 mg.
Promethazine Hydrochloride	12.5 mg.
Scopolamine Hydrobromide	.6 mg.

The pentobarbital sodium is administered orally while the remaining three agents are combined in a single syringe and injected by either the intravenous or intramuscular route. Rationale for the combination is predicated upon the fact that the action of the analgesic and barbiturate components is potentiated by the presence of promethazine hydrochloride. Similar potentiation of scopolamine hydrobromide does not take place. The combination, therefore, provides a sedative-analgesic effect achieved at one-half the dosage ordinarily required with these barbiturate and analgesic agents.

This premedication elevates the pain threshold. In addition tension and anxiety are relieved, thus predisposing to a more relaxed treatment session for both the patient and surgeon.[1] When signs and symptoms indicative of adequate drug activity are observed, injection of the local anesthetic is accomplished without anxiety or discomfort. The patient is then placed in a reclining position and the operation begun. Most patients fall into a light sleep and upon wakening retain no unpleasant memory associated with the operation. It is, of course, mandatory that patients who have been heavily sedated should be discharged only in the care of responsible persons. Patients should be observed until the predictable and apparent effects of the drug complex have been dissipated. Specific instructions concerning care of the patient during this period should be given to the person who assumes responsibility at time of discharge.

LOCAL ANESTHETICS

Lidocaine ointment is applied topically prior to needle puncture. This technic materially diminishes the initial pain of injec-

tion. Lidocaine combined with epinephrine, 1:100,000, is utilized for infiltration and block anesthesia.

POSTOPERATIVE MEDICATION

The dental profession has long recognized the need for a potent, non-narcotic drug which will provide adequate and prolonged analgesia during the postoperative period. Experience has indicated that in the management of pain following extensive periodontal surgery analgesic drugs combined with a sedative or mild tranquilizer frequently are more beneficial than agents providing analgesia only. We have found a new combination of an analgesic and a tranquilizing drug, plus acetylsalicylic acid, to be most satisfactory in meeting our postoperative analgesia requirements. The combination referred to comprises:

Dextro Propoxyphene Hydrochloride	32 mg.
Phenaglycodol	150 mg.
Acetylsalicylic acid	325 mg.

The preparation is available commercially as Darvo-Tran and will be so referred to for ease in discussion in the text to follow. The following comments concerning the major components of Darvo-Tran may be of interest to the reader not acquainted with the preparation.

Dextro propoxyphene hydrochloride (Darvon) is a relatively new non-narcotic analgesic agent[2] with a range of potency comparable to codeine. Extensive clinical studies with the agent, alone and in combination with acetylsalicylic acid-acetophenetidin-caffeine preparations have indicated the drug is effective and safe.[3,4]

Phenaglycodol (Ultran) is a mild tranquilizer. Loomis, *et al.*,[5] have demonstrated the agent in therapeutic doses does not adversely affect mental alertness or manual dexterity. The agent is, therefore, safe for use in ambulatory patients who must return to work, drive their cars, or engage in other occupational activities. Clinical studies have indicated that phenaglycodol provides an extensive range of application as an effective and safe tranquilizing agent.[6,7]

The present study incorporates observations on Darvo-Tran in approximately 300 patients who received the drug postoperatively. The professional judgment of the investigators and the subjective reactions reported by patients were used to determine the effectiveness of the agent during the postoperative period.

Before the Darvo-Tran study codeine plus acetylsalicylic acid had been used routinely for a period of about fifteen years. This combination provided effective pain control, but its use in many patients was accompanied by undesirable side-effects such as nausea, constipation, etc. Since Darvo-Tran became available for study, it has been administered routinely with the resultant discontinuation of the codeine-acetylsalicylic acid combination.

For the average patient one capsule of Darvo-Tran is prescribed three times daily and again at bedtime. Administration every four hours is suggested if pain is very severe. The regimen is continued for five to seven days depending upon individual analgesic requirements.

Approximately 90 percent of the patients participating in this study reported excellent analgesia without undesirable side-effects. The remaining 10 percent indicated that severe pain was not adequately controlled with Darvo-Tran. For this group substitute medication consisting of meperidine hydrochloride, 30 mg., and acetylsalicylic acid-acetophenetidin-caffeine compound was prescribed for the period when severe pain existed. Instructions were given to discontinue Darvo-Tran while taking the narcotic-containing preparation. Use of Darvo-Tran was resumed within twenty-four to forty-eight hours with good results.

CONCLUSIONS

1. Based on a study of approximately 300 patients undergoing periodontal surgery, it was found that Darvo-Tran pro-

vides excellent analgesia without the side-effects attending the use of narcotics.

2. The presence of phenaglycodol in the Darvo-Tran formula provides a desirable mild tranquilizing effect. In addition, the presence of phenaglycodol appears to enhance the analgesic effect of Darvon.

3. In our opinion Darvo-Tran is the analgesic-sedative agent of choice in ambulatory dental patients having undergone periodontal surgery.

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Book Review . . . .

History of Dentistry in Colorado. 1859-1959. By William A. Douglas. *Printed by Johnson Publishing Co., Boulder, Colorado, for the Historical Research Committee of the Colorado State Dental Association, 1959. 277 pages including index and illustrations. Price, $5.00.*

Readers interested in the history of dentistry will wish to read the interesting book on the history of dentistry in Colorado which has been released recently by the Historical Research Committee of the Colorado State Dental Association. The book was written by Mr. William A. Douglas, Assistant Registrar of the University of Colorado. Mr. Douglas obviously made a thorough study of early dentistry in Colorado and collected many interesting pictures which are reproduced in the book. Although some of the illustrations in the copy seen by the editor leave something to be desired, in general—the book is well organized and well written. It is interesting to note that on page 223 the author published a picture of the tooth brush drill which was held in Walzenberg, Colorado, in April 1917.

It is to be hoped that other states will become interested in publishing histories of the development of dentistry in their areas while many of those individuals who are responsible for the rapid development of dentistry are still alive. Every year makes it more difficult for an authentic history to be written.

A New Oral Adhesive-Protectant-Vehicle

by AUSTIN H. KUTSCHER, D.D.S., EDWARD V. ZEGARELLI, D.D.S., M.S., FRANK E. BEUBE, D.D.S., IRVING B. STERN, D.D.S., FERDINAND TUOTI, D.M.D., ROBERT E. HERLANDS, D.D.S., CHARLES L. BERMAN, D.D.S., AND JAMES L. MERCADANTE, D.D.S., *New York, New York*

THE useful properties of a new adhesive-vehicle preparation designed especially for the application of drugs to the oral mucous membranes have been previously reported.[1] It was found that this vehicle adhered to the site of application for periods varying from 15 minutes to two hours[1]—the variations dependent upon the degree of activity at the various sites, the "washing action" of saliva, and the amount of medication applied.

Since all materials placed in the mouth are generally swallowed, any systemic activity which a vehicle may possess must be considered. The components of this vehicle (gelatin, pectin, and carboxymethylcellulose in a mineral oil-polyethylene base) have all been found relatively free of deleterious, toxic or allergenic properties.[2-6] No evidences of irritation, side reactions, or toxicity have been observed either locally or systemically following single or repeated applications of the adhesive vehicle.[1]

During the course of investigations concerned with the use of drugs incorporated in this adhesive vehicle, another "action" of this preparation was noted. It was our experience that the adhesive vehicle, per se, contributed to the amelioration of certain lesions through its "action" as a local protectant.

Thus, this paper is concerned with the protectant action of this adhesive substance used in a variety of clinical circumstances. Frequently, the preparation was utilized in the same patient in more than one situation. When used as a placebo or as a protectant per se, approximately 50-250 mgm of the adhesive mixture were applied to the lesion under treatment, usually 4 times a day and particularly before retiring at night.

USE AS A VEHICLE

The adhesive substance described above has been used as a vehicle to carry various medications to oral lesions in: 5 patients with oral hyperkeratosis; 18 patients with recurrent ulcerative stomatitis; 4 patients with geographic tongue; 3 patients with idiopathic orolingual paresthesia; 7 patients with oral lichen planus; 17 patients with denture stomatitis; 9 patients with desquamative gingivitis or stomatitis; 3 patients with oral idiopathic chronic inflammation or ulceration; 3 patients with oral erythema multiforme; 14 patients with periodontal disease of various types; and following biopsy in 14 patients subjected to minor gingival surgery. Many of these patients (those with persistent, chronic, or recurrent lesions) had previously been observed, untreated, for significant periods or treated with various therapeutic agents including placebos and thus served as their own controls for the observations reported herewith.

Those patients who previously experienced subjective distress from their oral lesions uniformly reported unusually prompt subjective relief nearly immediately after application of the adhesive prepara-

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Divisions of Stomatology and Prosthetic Dentistry and Section of Periodontology, School of Dental and Oral Surgery, Columbia University, New York, New York, and the Institute of Cancer Research, College of Physicians and Surgeons, Columbia University, New York, New York.

The adhesive-protectant used in this study is available from E. R. Squibb and Sons as "Orabase" and was supplied by The Squibb Institute for Medical Research, New Brunswick, New Jersey.

The authors acknowledge the assistance of Richard Castoria, D.D.S.*, Philip Fenster, D.D.S.*, and David Hendell, D.D.S.*.

*National Institutes of Health Student Research Fellow.

tion. This relief, which was present only as long as the adhesive covered the lesion, appeared greater and more rapid in onset than that relief which could be attributed to the immediate action of the therapeutic agent incorporated in the vehicle.

USE AS A PROTECTANT OR PLACEBO PER SE

The adhesive substance administered alone as a "protectant" or as a "placebo" was studied as follows: in 6 patients with recurrent ulcerative stomatitis, 12 patients with idiopathic orolingual paresthesia, 6 patients with oral lichen planus, 3 patients with desquamative gingivitis, 2 patients with idiopathic chronic inflammation or ulceration, 2 patients with oral erythema multiforme, 5 patients with geographic tongue, 8 patients with hyperkeratosis, 12 patients with denture stomatitis, 12 patients with traumatic ulcers, 10 patients undergoing surgical periodontal therapy where portions of the usual periodontal pack were lost, 14 patients who were subjected to minor gingival surgery, and 12 patients where, for reasons of patient's discomfort or displeasure with the standard periodontal surgical pack, the pack was removed in 4 to 5 days.

Many of these patients (those with persistent, chronic, or recurrent lesions) had previously been observed, untreated, for significant periods or treated with various therapeutic agents including placebos and thus served as their own controls for the observations reported herewith.

Following application of the adhesive, most patients with recurrent ulcerative stomatitis, idiopathic orolingual paresthesia, oral lichen planus, traumatic ulcers, desquamative gingivitis or stomatitis, idiopathic chronic inflammation, and oral erythema multiforme reported considerable relief of symptomatic discomfort ("diminished pain and/or discomfort," "decreased soreness," and "increased comfort"). Patients with oral hyperkeratosis, geographic tongue, and denture stomatitis—diseases which are generally asymptomatic—did not report any changes.

Objectively, amelioration of mild, small keratotic lesions was noted in 5 of the 8 selected cases covered repeatedly during the day with the vehicle. These effects were considered to be largely the result of the protection afforded the lesion from a mild chronic irritant.

Although improvement was noted in 3 patients with idiopathic orolingual paresthesia, it was concluded that the improvement was largely either a placebo effect or a protectant action against a "toying," "testing," or rubbing action of the tongue against the teeth.

The application of the adhesive vehicle in liberal quantities (500 mgm) over minor gingival surgery or biopsy sites resulted in a considerable diminution of pain, soreness, and distress as compared to that usually experienced by patients similarly treated surgically but without the subsequent application of the adhesive. Repeated (4 or more times a day) application of the preparation (by finger—without the use of a confining covering such as a denture base or adhesive foil) was necessary. Following the premature loss of periodontal pack, use of the adhesive provided suitable protection for the denuded areas according to the patients' subjective comments. Again, however, repeated (4 or more times a day) application of the preparation was necessary. Where the adhesive protectant was used to replace the standard periodontal pack after 4 to 5 days owing to patient's discomfort or displeasure, the vehicle subsequently made an adequate substitute for the pack as evidenced by patients' subjective symptoms and clinical observations of healing progress.

The following additional observations with regard to findings derived from Parts I and II seem pertinent. The duration of symptomatic relief, where experienced, paralleled fully the time during which the protectant remained in contact with and as a covering over the lesion. No objective changes attributable to the use of the ad-

hesive protectant were noted except for those described previously. No evidences of side reactions, toxicity, or allergy were noted except in one patient with multiple allergic tendencies. In this patient, one or a few giant hives appeared (on the scalp and back of the neck) on several occasions following the application of as little as 60 mgm of the unmedicated adhesive formulation.

DISCUSSION

The ameliorative effects resultant from the use of the preparation as described above indicate that this formulation exerts its influence (1) by protecting oral lesions from external environmental irritants and (2) by allowing natural healing processes to proceed undisturbed in spite of the continued presence of such irritants.

Such a vehicle-protectant action may thus be indicated for adjunctive care in the treatment of the oral lesions mentioned above.

SUMMARY

The use of a preparation containing gelatin, pectin, and carboxymethylcellulose in a mineral oil-polyethylene base for its adherent protectant properties in the mouth is described.

Considerable symptomatic relief and occasional instances of objective improvement were noted in patients with various oral soft tissue lesions when the latter were covered with the protectant vehicle. The duration of subjective symptomatic relief was restricted to and paralleled the period during which the protectant remained over the lesion.

630 West 168th Street
New York 32, New York

The authors acknowledge the appreciated assistance of Mrs. Aldonna Skislak, Mrs. Helene W. Kutscher and Mrs. Barbara Slaughter.

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GREATER MIAMI CHAPTER OF ALPHA OMEGA FRATERNITY

The second Annual Sunshine Seminar will be presented by the Alumni of the Greater Miami Chapter of Alpha Omega Fraternity at the Deauville Hotel, Miami Beach, Florida, January 22-25, 1960. Clinicians include Drs. Lewis Fox, Saul Schluger, Herbert J. Bartelstone and Leonard Hirschfeld. The fee for the seminar is $150.

For further information write Dr. Irving Gordon, 420 Lincoln Road, Miami Beach 39, Florida.

Clinical Evaluation of an Oxygen Liberating Preparation Used Adjunctively in Root Planning

by PATRICK D. TOTO, D.D.S., M.S., MALBURN WILDERMAN, D.D.S., M.S. AND CHARLES REEVE, D.D.S.,* *Chicago, Illinois*

THE use of oxygen liberating compounds for the treatment of gingival disease has been used by dentists and physicians for a long time. The nature of the usefulness of such preparations generally was based on the antiseptic property, particularly against pathogenic anaerobes. Also, oxygen has been applied directly to the gingiva by insufflation and in high concentration, thirty percent, as Superoxol. In such instances it was observed that inflamed gingiva resolved bringing about a clinically normal state.

A currently available oxygen liberating compound, Amosan,** was used in a dou-

*The departments of Oral Pathology and Periodontics.

**Amosan was kindly furnished by the Knox Company, 1400 Cohuenga, Los Angeles 28, California.

TABLE I—CODE #70

Case Number	*Duration*	*P.M.A. Start*	*P.M.A. Finish*	*% Decrease in Inflammation*	*Average Decrease*
1.	One week	13	5	61.5%	
2.	" "	6	4	33.4%	36.1%
3.	" "	22	19	13.5%	
4.	Two weeks	24	8	66.7%	
5.	" "	17	2	88.2%	
6.	" "	22	1	95.5%	
7.	" "	32	18	43.7%	
8.	" "	12	9	25.0%	
9.	" "	25	8	68.0%	60.7%
10.	" "	21	8	61.9%	
11.	" "	18	6	66.7%	
12.	" "	21	15	28.5%	
13.	" "	8	3	62.5%	
14.	Three weeks	19	3	84.2%	
15.	" "	39	25	35.9%	
16.	" "	47	13	72.3%	
17.	" "	25	0	100.0%	
18.	" "	6	0	100.0%	78.3%
19.	" "	34	12	64.7%	
20.	" "	53	16	69.8%	
21.	" "	10	0	100.0%	
22.	Four weeks	41	5	87.8%	
23.	" "	5	0	100.0%	
24.	" "	16	2	87.5%	80.3%
25.	" "	39	21	46.1%	
26.	Five weeks	30	4	86.7%	86.7%

Average Duration	*Average Decrease in Inflammation*
One week	36.1%
Two weeks	60.7%
Three weeks	78.3%
Four weeks	80.3%

ble blind study to determine its usefulness in the treatment of periodontal disease.

The study of the value of Amosan as adjunctive therapy in root planning was conducted using sixty-five subjects whose age ranged from twenty to sixty-five years. Such selected subjects all were diagnosed as chronic periodontitis. The treatment in each case was root planning and polishing the tooth surfaces.

The subjects were equally divided. One-half of the group was given weekly supplies of twenty-one packets of code #70,* and the other half code #80.** The instructions were to rinse the mouth using a tablespoon of water with each packet, three times each day after brushing their teeth and gingival massage. The subjects returned at weekly intervals for P.M.A. evaluation and treatment. The duration of treatment ranged from one week to five weeks. Fifty-three of the sixty-five subjects completed the period of treatment and observation.

*Code #70 Placebo. **Code #80 Amosan.

TABLE II—CODE #80

Case Number	*Duration*	*P.M.A. Start*	*P.M.A. Finish*	*% Decrease in Inflammation*	*Average Decrease*
1.	Four days	12	12	0	0.0%
2.	One week	14	1	92.8%	
3.	" "	6	2	66.7%	55.0%
4.	" "	36	34	5.5%	
5.	Two weeks	26	20	22.0%	
6.	" "	43	2	95.4%	
7.	" "	5	0	100.0%	
8.	" "	9	0	100.0%	
9.	" "	12	3	75.0%	72.8%
10.	" "	11	4	63.7%	
11.	" "	20	6	70.0%	
12.	" "	23	10	56.5%	
13.	Three weeks	28	5	82.1%	
14.	" "	45	7	84.5%	
15.	" "	22	2	90.9%	
16.	" "	12	0	100.0%	
17.	" "	25	1	96.0%	
18.	" "	26	5	80.8%	82.4%
19.	" "	7	2	71.4%	
20.	" "	9	0	100.0%	
21.	" "	11	2	81.8%	
22.	" "	37	9	75.7%	
23.	" "	25	14	44.0%	
24.	Four weeks	43	13	69.8%	
25.	" "	10	0	100.0%	89.9%
26.	" "	12	0	100.0%	
27.	Five weeks	19	9	53.7%	53.7%

Average Duration	*Average Decrease in Inflammation*
One week	55.0%
Two weeks	72.8%
Three weeks	82.4%
Four weeks	89.9%

In each case, the number of inflamed papilla marginal and attached gingiva was counted and recorded at the beginning of the experimental period. This continued with each visit and terminated with their treatment. The number of inflamed P.M.A. in each case was estimated at one hundred percent at the initial examination. At the terminal examination, the number of inflamed P.M.A. similarly was reduced proportionately to a percentage figure. Thus, in each case the number of P.M.A. at the start and finish of each was recorded with the estimated percentage reduction in inflammation. Finally, the averages of the percentage reduction was estimated for each period of one, two, three, four, and five weeks. The results of such findings are tabulated in Table I, and Table II.

It is interesting that as the duration of treatment increased from one week to five weeks, the percentage decrease in inflammation increases both in cases using #70 and #80. This must be attributed to the scaling and polishing of the teeth. On the other hand, the differences in reduction of inflammation in any period shows that the subjects on #80 show a greater reduction than the subjects using #70. At the end of one week, there was 18.9% greater decrease of inflammation using #80; at two weeks, 12.1%; at three weeks, 4.1%; four weeks, 9.6%. Such differences in percentages can be attributed to any active agent in the rinse. This must exclude the physical activity of oral rinse, since both #70 and #80 were used this way.

It can be concluded that #80 increased the reduction of inflammation over simple root planning in given weekly periods by 18.9%, 12.1%, 4.1%, and 9.6%.

The decrease in the inflammation percentage differences as the duration of the treatment increased must be attributed to the scaling procedure. On the other hand, the higher percentage of decrease in inflammation in the cases using #80 can be attributed to the action of its properties.

TUFTS UNIVERSITY SCHOOL OF DENTAL MEDICINE

Tufts University School of Dental Medicine announces the following postgraduate refresher course:

DPG. 602—CLINICAL ADJUSTMENT OF THE OCCLUSION

March 14, 15, 16, 1960

by DR. BERNARD JANKELSON, CLINICAL ASSOCIATE AND LECTURER, UNIVERSITY OF WASHINGTON SCHOOL OF DENTISTRY

The first day will be devoted to clarification of the physiology of the Stomatognathic area, particularly as it pertains to the occlusion of the teeth. It will be shown that as the result of this information our concepts of the basic nature of the dentition and of occlusion must be considerably modified. Tuition $100.00—Class Limited.

The remainder of the course will demonstrate the clinical adjustment of a series of cases by means of television projection. The clinical diagnosis and treatment will be done directly in the mouth. A sufficient number of cases will be done to cover the various conditions encountered in practice.

AMERICAN ACADEMY OF PERIODONTOLOGY

Spring Conference

April 25, 26, 27, 1960

Monday, April 25

8:00 a.m. - 9:00 a.m.	Registration, 2004 Kellogg Bldg.
9:30 a.m. - 12:00 noon	"Interrelation of Medicine and Dentistry in the Field of Infectious Diseases" Edward L. Quinn, M.D., Chief, Division of Infectious Diseases, Henry Ford Hospital, Detroit.
2:00 p.m. - 3:15 p.m.	"The Dentist and Cardiac Disease" William L. Morgan, M.D., Associate, Division of Cardiovascular Diseases, Henry Ford Hospital, Detroit.
3:15 p.m. - 4:30 p.m.	"The Dentist and the Cardiac Patient" John W. Keyes, M.D., Chief, Division of Cardiovascular Diseases, Henry Ford Hospital, Detroit.

Tuesday, April 26

9:00 a.m. - 12:00 noon	"The Dermatology Patient and the Periodontist" Robert E. Burns, M.D., Associate, Division of Dermatology, Henry Ford Hospital, Detroit.
1:30 p.m. - 4:00 p.m.	"Recent Research in Periodontal Disease" Helmut A. Zander, D.D.S., Eastman Dental Dispensary, Rochester, New York.
6:00 p.m.	Social Hour and Banquet—Michigan Union. Speaker: To be announced

Wednesday, April 27

9:00 a.m. - 12:00 noon and 1:45 p.m. - 3:30 p.m.	"Concepts of Occlusion" (Including: Clinical and electromyographic investigation of occlusal and temporomandibular joint disorders) Sigurd Ramfjord, L.D.S., M.S., Ph.D. Professor of Dentistry (Periodontics) University of Michigan School of Dentistry.

WESTERN SOCIETY OF PERIODONTOLOGY

The Western Society of Periodontology announces that its Seventh Annual Las Vegas Meeting will be held March 27, 28 and 29, 1960. The speaker will be Dr. Hans Selye.

For further information write Dr. Howard A. Tyrell, 1508 North Sycamore Street, Santa Ana, California.

Editorial . . . .

Advancement of the understanding of the etiology of dental calculus is progressing slowly. The relationship between food intake and calculus formation has long been suspected; G. V. Black reported that calculus was deposited after the "ingestion of an excessive amount of almost any food." (Black, G. V., A Work on Special Dental Pathology, 3rd ed., Medico Dental Pub. Co., Chicago, 1924, p. 86.) This is a "lead" that we have always thought worthy of study. In this issue of the *Journal* the lead article by Stewart and Burnett describes some interesting studies concerning attempts to produce salivary calculus experimentally in small laboratory animals, and an evaluation of some factors which affect its rate of deposition. Such studies of course are not new. For example, King (J. Dent. Res. 34:650, 1955) and his associates have produced material they have identified by X-ray diffraction as being similar to human salivary calculus in both the hamster and ferret by feeding the animals a calculus-producing diet. Calculus has also been found to occur in dogs, cats, horses, mules, pigs, the elephant, bear and many other animals, although the similarity to human calculus in all these species has not been shown.

In the study by Stewart and Burnett reported in this issue, no evidence was provided to show that the material they state to be calcified was in fact similar to human calculus, but in all probability it did have at least certain similarities. It is of interest that Stewart and Burnett have shown that the use of dehydrated blood serum and peanuts is quite effective in producing the matrix apparently needed for subsequent deposition of inorganic material.

As King has repeatedly pointed out, one of the main differences between calculus found in the small laboratory animals and humans is the degree of calcification. The report of Stewart and Burnett suggests the same since the diet they used was effective in forming plaques but not always were these plaques calcified. These studies are of real importance in preparing for the development of anti-calculus substances. The recently-reported clinical study by Jenson (J.A.D.A. 59:923, 1959) using a dentifrice containing dehydrated pancreas for reducing calculus formation is encouraging; the development of a satisfactory means of controlling calculus formation in the human seems imminent.

Of particular interest in the work of Stewart and Burnett is the finding that sodium bicarbonate has a tendency to produce more calculus than was found in any of the other groups studied. Similar conclusions also seem well founded in the controlled observation of clinicians who note the increased deposition of calculus in patients who through choice use substances which increase the alkalinity of the mouth. No reports are known of adequate clinical tests on the use of baking soda as a dentifrice. One wonders if the dental profession should continue its endorsement of baking soda as a tooth cleansing agent, at least until good clinical tests have been reported which evaluate its effectiveness, and its effects, when used for long periods of time.

PRESIDENTIAL ADDRESS—1959

by JOSEPH L. BERNIER, D.D.S., M.S., F.D.S., R.C.S. *(Eng.)*

Occasionally the concerted efforts of men produce a result so useful that the sometime questioned wisdom of man is redeemed in the eyes of all. The American Academy of Periodontology is such an accomplishment. To have been the presiding officer during the forty-fifth year of its activities and during that period signalling the one-hundredth anniversary of the American Dental Association was indeed a rare privilege. Only one person could have enjoyed that great distinction and that it should have been me makes me humble of heart.

This, then, is the forty-fifth annual meeting and I welcome you most cordially. You will, I feel certain, be intellectually stimulated and rewarded.

I offer a special word of welcome to our many colleagues from foreign countries who join us in this scientific assembly and who will share with us their knowledge and experiences. This beyond all doubt will be a most worth-while experience for all.

In many ways this has been an unusual year. Because of the presence of our foreign guests and because of the Centennial of the American Dental Association, every effort has been made to have an outstanding meeting. This was a lofty objective in view of the superior meetings of the past. I think you will like the program and I know that the color television presentation will be a memorable event. We are the first specialty group to use this medium at a national meeting in this dimension and scope. This is a time when your serving president may present his views on the past, present and future of our Academy. The past and present are the yardsticks which help us measure and determine our future actions and it is upon such a template I wish to discuss some segments of the future as I view them and what we must do to be compatible with the inevitability of change.

On July 9th and 10th of this year the Council on Dental Education of the American Dental Association invited representatives of the special areas of dentistry, and others interested in the complicated problem of specialization, to meet with them in Chicago. I shall not bore you with the many actions taken by the American Dental Association regarding specialty boards previous to that time since they are, no doubt, familiar to all of you. The most important is the moratorium on new specialty groups which has been in force for several years. At this meeting the current report of the Council on Dental Education to be presented to the House of Delegates next week was discussed.

In this report the council expresses its opinion and recommends adoption of certain resolutions on the following matters:

a) Statement of Policy on Specialists and Specialties in dentistry.

b) Requirements for National Certifying Boards for special areas of dental practice.

Certain points in the former category should be discussed. I refer to the following which are quoted verbatim:

"Specialization by nature and definition demands full time or exclusive attention from the specialist" and "A practitioner may have every qualification of the specialist but as long as he does not hold himself out to the public as a specialist, he does not expose himself to legal, professional and ethical sanctions."

Upon a clear and unemotional analysis of these statements one comes to the logical

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conclusion that on the basis of their ethical properness they offer little room for dispute and will, in my opinion, be adopted by the House of Delegates of the American Dental Association. Thus will the stage be set for determining the administrative status of a specialist.

Certain of the specialty areas in dentistry, of which periodontology is one, must now urgently and critically appraise the dimension of their sphere of practice so that these policies may be fairly and logically applied. There seems to be ample reason to believe that the Council on Dental Education will leave to the organizations representing specialty areas, the matter of clearly defining the extent and limitations of their specialty practices. This, in the forty-five years of its existence, the American Academy of Periodontology has not done—it must do so now.

Most important in this regard is the expressed view of the council on Dental Education that it recognizes that there will be overlapping between specialty areas, one with another and to some extent with the practice of general dentistry. This too, in my opinion, is logical, and should make it a relatively simple matter to define our area of dental practice.

It is important that we define the practice of periodontology in lucid terms without regard to what definition other specialty groups may give to their area of practice. In those areas where there is overlapping, those best qualified will survive.

Having done this we must then look to our members to appraise their own practice in the light of such a definition. The high degree of ethics known to exist in this academy leaves no doubt as to the propriety of this procedure. There can be no question of sanctions against anyone except those which he himself imposes.

With such a program we can assure ourselves, organized dentistry and the public at large of our compliance with the rules of ethics which are, of course, the natural will of the majority.

To this end I therefore recommend to the Executive Council that it appoint a special committee to draft a definition outlining the scope of the practice of periodontology to be presented for approval of the general assembly at the next annual session and that it (the council) invite the American Board of Periodontology to representation on this special committee.

Further, I recommend that this same special committee review the scientific requirements for certification in periodontology to determine their suitability in terms of their proposed definition.

We may all be assured that there is more to come with regard to the matter of properly orienting the specialty groups and areas. However, it would appear that for the moment if we will do our very best to understand the views of the council on dental education in this matter, that we will be indeed discharging our responsibility. There is to be another meeting of representatives of the specialty boards and organizations to be called by the council on dental education in the spring of 1960. At this time certain groupings of the specialty areas will be suggested. We would be well advised to have knowledge of these proposals and to face this inevitability with a desire to be cooperative, thus achieving what we know to be best for periodontology.

Now I wish to leave that particular matter with you for your earnest deliberations and pass on to a discussion of some of the accomplishments of our group which I think from time to time should be given the weight they deserve. It is a somewhat natural tendency to place much emphasis upon the things we feel need doing without occasionally enjoying the pleasant reflections of a successful past which is the heritage of this academy.

In its 45 years of existence, the American Academy of Periodontology has become one of the outstanding specialty groups in dentistry. When it speaks on matters which properly concern its members, its voice has authority. This could only have happened as a result of sound, careful, methodical actions throughout the years. Having searched diligently through reports and records of our academy's actions, which were available to me, I am truly amazed at the accomplishments which have been recorded. When one considers the time and effort that has been spent in the field of classification and nomenclature, or the efforts this organization has made to properly reflect its position in the matter of the undergraduate and graduate teaching of this subject, and when one further reflects on the influence these activities have had in the development of our specialty it is difficult to be other than extremely proud.

In July of this year there came to me the publication of the "Proceedings of a Workshop for Teachers in Periodontology." This beautifully bound volume is a tribute to the Academy and to Dr. Harold G. Ray who was its moving spirit. I was unable to be at that meeting, but I have read the proceedings in detail, and while I have some misgivings over the fact that many of the viewpoints and expressions of those present seem to be couched in the language and thinking of the past, I still feel that this was an extremely important effort. I hope you will permit a bit of editorializing at this point. After reading the report the thought occurred to me that we would all do well to again satisfy ourselves on the important distinction between rhetoric and logic. So many reports, and these proceedings are no exception, seem to be predicated on the need for convincing someone of a point of view and to use for this purpose a time honored art of rhetoric rather than the precise method of logic. There is really no need to continually present concepts and ideas in the words, paragraphs and phrases of others. It is quite possible to avoid stereotyped expressions of the past, and in so doing to make it possible to view the problem from a new point of vantage. Such a practice is, to say the least, intellectually stimulating.

I was particularly interested in the comments of one participant who felt dismayed at the lack of progress in periodontics chiefly because the cause of periodontal disease has not yet been described and delineated in detail. Such constant appeals to find the cause of periodontal disease makes one speculate on the erudition of those who continually make such demands. There is no question as to the desirability of knowing this but to suggest that until it is known that no substantial progress has been made is not realistic. There are biologic processes in the human organism which because of improper interplay may produce clinical phenomena which in the strict sense of the word defy etiology. This should not be a matter of great concern since the proper management of disease, indeed even its elimination, are distinctly possible without "discovering" a precise etiology. Take dental caries, for instance, are all concerned with the disease in agreement as to its precise etiology? I think not. Is fluoridation of water, the most effective means of controlling this disease, based on a knowledge of the etiology of dental caries? Certainly not. Nevertheless we can all agree that the "workshop" was a monumental task of which the Academy should be proud.

This is not a time for the president to speak at great length. He can, as I have done, take comfort in the accomplishments of others but he cannot rightfully do so without an acknowledgment to those who in essence have done the work. To all who have served on committees during the past year, I am extremely grateful. Dr. B. O. A. Thomas, Dr. Irving Glickman, Dr. Craft Hopper, and others have displayed a magnificent desire to make this one of the outstanding meetings of the Academy—they have done so. The hub of the organization, our office in Peoria, is not only a physical point about which all of our activities revolve, it is the source of wisdom which is essential to the proper functioning of our organization. Over the many years that Dr. Clarke Chamberlain and his able

helper, Mrs. Lorrie Hill, have been the hub of this wheel, we have progressed remarkably. This last year has been no exception. Indeed without them this meeting would not have come to pass. To both of them I extend my very special thanks. Each year reference is made to the ability, the thoroughness and the forthrightness with which our able treasurer Dr. Aiguir discharges his duties. I need hardly point out to you that this Academy is indeed a major portion of his life. He is dedicated to his responsibilities and with such a person we could no nothing other than prosper. To him I am grateful. Many others deserve special commendation and I feel certain that each and every one of them will accept my very special tribute for the cooperative way in which they have helped during the year.

As a final word, I would like to make mention of our banquet to be held on Friday night at the Essex House. This is a special event. Several of our distinguished members will be properly honored and the event will be noteworthy, because of these acknowledgments. You will, I am sure, have a most pleasant evening. May I then, in conclusion, say to you that you could not have done me a greater honor than to permit me to serve as your presiding officer during this year. It has been a memorable experience and one which I shall long remember. Like most of you, I have been associated with many organizations and have been active with them in their deliberations. But in none, has the spirit of gentlemanly congeniality been displayed to a greater extent than in the American Academy of Periodontology. I rejoice, therefore, that this experience has come my way and I hope sincerely that all of the advantages of this association are not mine to tuck into my recollections of the past; for enjoyment in the years to come.

REPORT OF THE COMMITTEE ON PRESIDENT'S REPORT

The Committee on the President's report has carefully reviewed the thoughtful address which President Bernier delivered in his usual eloquent manner at the opening session of this meeting.

The Committee believes that problems associated with the control of dental specialties are critical and concurs with President Bernier's advice that members of the Academy must give close attention to developments in this field. It urges support to his recommendation that the Executive Council appoint a special committee to formulate the definition which delineates the scope of practice of periodontology, to review scientific requirements for certification in periodontology and to be prepared to present the attitudes of the Academy of Periodontology at meetings called by the Council on Dental Education of the American Dental Association.

The Committee agrees with the President's attitude that even though final chapters have not been written on the etiology of periodontal diseases, much has been learned. It is untrue to imply that all the answers have been found for problems in periodontology, but it is equally untrue that no progress is being made. The Committee agrees with President Bernier that the workshop with the teachers in periodontology was one of which we can be proud and encourages Dr. Harold Ray and his committee to continue their plans for another workshop.

The Committee recognizes that the contributions of the Academy of Periodontology are dependent upon the dedicated services of "volunteer workers." The Committee agrees that members of the various committees who have had the responsibilities of the details

of this meeting deserve the Academy's commendation. To the list given by the President, the Committee insists upon adding the name of Doctor Bernier. The Academy has enjoyed a successful year and Dr. Bernier deserves much of the credit for it.

Respectfully submitted,
Stanley Baker
M. K. Hine
Donald Kerr, Chairman

REPORT OF A.A.P. EDUCATION COMMITTEE

In recent years there has been an ever increasing demand by dentists for more information in periodontology. Local dental societies, large dental organizations, and dental schools throughout the country have reported that requests for periodontia lectures and courses surpass that for most dental subjects. Each year at the annual meeting of the American Academy of Periodontology approximately two hundred non-member dentists, most of whom are general practitioners, attend the scientific sessions. One of the principal reasons for the development of this widespread interest in periodontology, is an awareness on the part of many dentists that in spite of what seemed proper treatment planning and the rendering of good services to their patients in operative dentistry, crown and bridge, endodontic therapy and partial dentures, many of their cases showed breakdown of the periodontal structures. These dentists discovered, although several years subsequent to graduation, through discussions with their colleagues, reading the literature and listening to lectures by periodontists at dental meetings, that it was no longer tenable to place the entire responsibility of failure of their cases, which were primarily treated with restorative dentistry, on poor periodontal tissue resistance or systemic factors. They also learned that periodontal care was an essential phase of a complete dental service and that extraction of teeth was not the only means of eradicating periodontal disease. Additional important and substantial information that dentists obtained, after consulting periodontists about periodontal problems in respect to their patients, was that the periodontal tissue destruction of these cases might have been prevented and that the etiology in most instances was known. They further learned that when these same patients were treated, their periodontal structures including the bone, showed repair.

It is a credit to progressive and alert dentists that through their own initiative they have accumulated a working knowledge of periodontology after receiving their dental degree. Dental practitioners should always continue their studies and take postgraduate courses, but a foundation in periodontology should be given at the undergraduate level. It has been shown through replies of a questionnaire which was mailed to all dental schools in the United States and Canada, by the Committee on Education of the American Academy of Periodontology, that the majority of these schools has set aside insufficient number of hours for teaching periodontology and the subject content in this major course was also inadequate. Lacking instruction in the fundamentals of periodontics, the recent dental graduate will naturally flounder as he attempts treating his patients. This will result in deficiencies of prescription and ultimate damage of the supporting structures of the teeth. The majority of dental students are not only deprived of basic training in periodontology but their knowledge of this highly important subject is not determined by Dental State Board examinations. Very few Boards require practical tests in this field of

study. Yet all patients who seek dental care require as the minimum, instruction in oral hygiene procedures for home use and oral prophylactic treatments in the office. Both of which contribute to a large degree towards the prevention of periodontal diseases.

SUBJECT MATTER IN UNDERGRADUATE PERIODONTOLOGY

It would seem obvious and imperative that the degree of Doctor of Dental Surgery should not be bestowed upon anyone until the individual has been trained in the fundamentals of all phases of dentistry including periodontology. It was pointed out in a recent paper[1] "Survey of Subject Matter Necessary For Undergraduate Experience In Periodontology," that: "It would be equally difficult to present to undergraduate students all of the documented information on periodontics as it would be for other courses in the dental curriculum. This level of advanced instruction should be reserved for postgraduate teaching. Specifically, students should receive basic instruction in the following:"

A. LECTURE AND SEMINAR SESSIONS:

1. Periodontal anatomy, histology and physiology
2. The form, position and occlusion of the teeth as related to the periodontium
3. Clinical periodontal pathology
4. Histopathology of the periodontium
5. Etiology of periodontal diseases
6. Periodontal examination procedures and diagnosis
7. Periodontal treatment technics and their rationale
8. Provisional and final fixed periodontal splinting
9. Periodontal prognosis
10. Treatment planning of cases with periodontal disease and occlusal problems.

B. CLINICAL TRAINING IN THE FOLLOWING TREATMENT METHODS:

1. Coronal scaling and polishing
2. Root and soft tissue curettage
3. Instruction in oral hygiene
4. Wire ligation and other temporary stabilizers
5. Selective tooth grinding
6. Basic periodontal surgery

CURRICULUM TIME FOR UNDERGRADUATE PERIODONTOLOGY:

The faculty of any dental school would, in general, accept the suggestions of their periodontia teaching staff in respect to the necessary periodontal subjects that should be taught to the undergraduate students. Probably most dental faculties would also not reject the fact that the retention of the natural dentition is dependent upon a healthy periodontium. Since this can be achieved through periodontal preventive measures, periodontology then becomes one of the major dental courses to be given at the undergraduate level and sufficient number of hours should be allotted for presenting basic information. Twenty, eighty, or one hundred hours during four years of undergraduate dental training, is far from adequate. Yet many schools are limited to these abbreviated teaching periods according to a survey by the Education Committee of the American Academy of Periodontology. Craig[2] pointed out that eighty-three hours was the average available time for teaching periodontics. Any increase in curriculum time for one dental course must of necessity reduce the number of hours for one or more other courses that are being taught, and it is this possible reduction of teaching time, especially in the area of restorative

dentistry, which dental faculties oppose. This is justifiable but not realistic. There is a phase familiar to everyone and appropriate for our purpose: "You cannot have your cake and eat it too." If it is essential to include the fundamentals of periodontology in the dental curriculum, then it will be necessary that dental educators review their curricula and objectively determine what modifications should be made in order to provide a balance in all dental courses. In the recently published book "Proceedings of a Workshop for Teachers in Periodontology,"[3] sponsored by the American Academy of Periodontology, the deliberations of the Committee on Curriculum Time and Sequence proposed the following: "In order to meet these objectives it is recommended that not less than 10 percent of the total curriculum time for the four years be allotted to periodontology. With an approximate 4,367 total hours in the dental curriculum, 10 percent or 440 hours would be available to teach periodontology adequately." The source of this time is not the sole concern of the departments of periodontology but should be shared by all dental educators. With the changing concepts of dentistry, it is incumbent upon dental schools to reevaluate their curricula content and time towards this end.

Respectfully Submitted,
C. Kenneth Collings
Henry M. Swenson
Frank E. Beube, Chairman

REFERENCES

1. Beube, F. E.: Survey of Subject Matter Necessary For Undergraduate Experience In Periodontology; Jl. of Dent. Educ., pp. 156-161, March 1958.

2. Craig, C. A.: Student Quantitative Clinical Achievement; Report of a Survey, Jl. of Dent. Educ., 16:7, 1952.

3. "Proceedings of Workshop For Teachers in Periodontology," University of California Printing Department, 1959.

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Spring Conference (1961)
Joseph Barkley, *Chairman*
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Some participants in the 1959 Meeting of the American Academy of Periodontology, New York City:

1. A.A.P. President Joseph L. Bernier, Washington, D. C.
2. President-elect James Hollers, San Antonio, Texas.
3. Dr. W. G. Cross, London, England.
4. Dr. Perry Ratcliff, San Bernardino, California, talking to Dr. Clarke Chamberlain, Peoria, Illinois, Secretary.
5. Dr. Frank Beube, New York City.
6. Dr. Harold Ray, Past President, San Francisco, California.
7. Dr. F. A. Carranza, Sr., Buenos Aires, Argentina, South America.
8. Dr. Arthur J. Held, Geneva, Switzerland.
9. Dr. Sigurd Ramfjord, Ann Arbor, Michigan.
10. Dr. James Aiguier, Treasurer, Philadelphia, Pennsylvania.

Members of the Council of the American Academy of Periodontology:

Left to right, first row: Dr. Clarke Chamberlain, Secretary, Dr. James Hollers, President, Dr. Joseph L. Bernier, Immediate Past President, Dr. James Aiguier, Treasurer, Mrs. Lorrie Hill, Secretary.

Second row: Dr. C. H. M. Williams, Dr. Richard Chace, Dr. Delbert Nachazel, Dr. E. A. Archer.

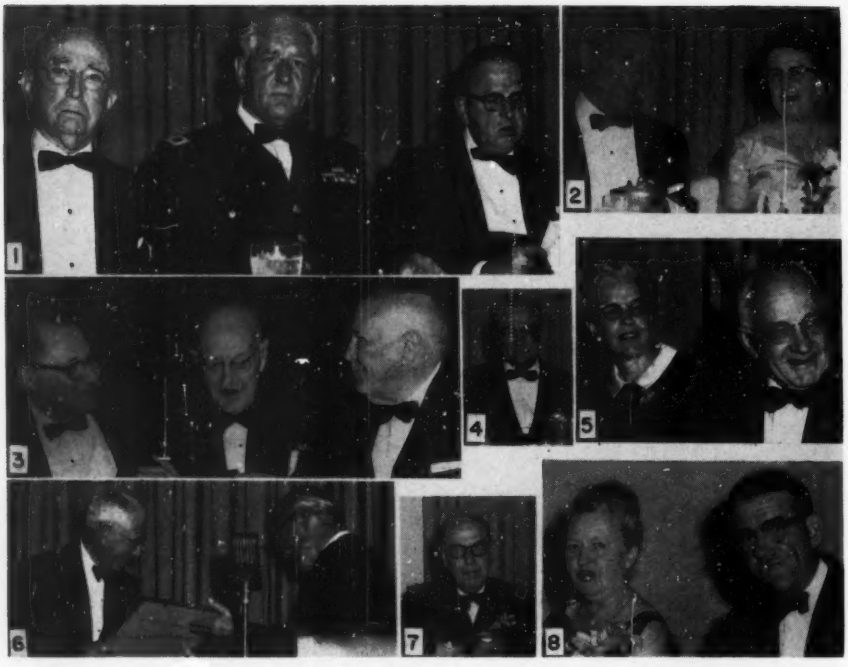
Not present: Dr. B. O. A. Thomas (President-elect), Dr. Frank E. Beube, and Dr. Craft A. Hopper.

ANNUAL COURSE AT AFIP

March 14-18, 1960

The Dental and Oral Pathology Division of the Armed Forces Institute of Pathology, Washington, D.C., announces its annual course entitled "Pathology of the Oral Regions." This course will be presented during the period March 14-18, 1960.

The substance of the course will be directed at the biologic aspects of general dental practice. Attendance is limited to one hundred and requests to attend should be made immediately since registration is very heavy. Members of the military establishments should make application in accordance with Circular No. 621-31, Department of the Army, dated 10 June 1959. For additional information please write J. L. Bernier, Colonel, D.C., U.S.A. Armed Forces Institute of Pathology, Washington 25, D.C.



Among those attending the annual dinner of the American Academy of Periodontology were:

1. Guest of Honor Dr. Arthur Merritt, New York, President Joseph L. Bernier and Dr. Norman Vincent Peale, speaker.
2. Dr. James Hollers and Mrs. Clarke Chamberlain.
3. Dr. Irving Glickman, Dr. Justin Towner and Dr. Olin Kirkland.
4. Dr. Raymond E. Johnson.
5. Mrs. M. K. Hine and Dr. Clarke Chamberlain.
6. Dr. Harold Leonard accepting the Fellowship Certificate from Dr. Harold Ray.
7. General Epperly, U.S. Army Dental Corps.
8. Mrs. Raymond E. Johnson and Dr. Donald Kerr.

"SEVEN AGES OF MAN" TO FEATURE ANNUAL MEETING OF AMERICAN ACADEMY OF DENTAL MEDICINE

The spotlight of scientific study will focus on "The Seven Ages of Man" at the 14th annual meeting of the American Academy of Dental Medicine, to be held May 27-30, 1960, at the Warwick Hotel, Philadelphia, it has been announced by Drs. Jacoby T. Rothner and Bernard B. Saturen, co-chairmen.

Inquiries concerning the 1960 annual meeting may be addressed to Dr. Rothner at Medical Arts Building, Philadelphia 2, or Dr. Saturen at 1930 Chestnut St., Philadelphia 3, Pa.

BOSTON UNIVERSITY

The Department of Stomatology, Division of Graduate Studies, School of Medicine, Boston University anonunces the following postgraduate courses:

Advanced Course in Periodontology, Dr. Henry M. Goldman, Dr. Bernard S. Chaikin, Dr. Gerald M. Kramer, Dr. William Pendergast, and Dr. Jack Bloom. March 14-19, 1960.

Periodontal Therapy, Dr. Henry M. Goldman, Dr. Bernard S. Chaikin, Dr. Gerald M. Kramer, Dr. William Pendergast, Dr. Jack Bloom, and Associates. April 4-9, 1960.

Periodontal Pathology, Dr. Henry M. Goldman and Dr. Jack Bloom. April 25-30, 1960.

Periodontal Prosthesis, Dr. Leo Talkov, Dr. David J. Baraban, Dr. Henry M. Goldman, Dr. Bernard S. Chaikin, and Dr. Gerald M. Kramer. May 16-20, 1960.

Surgical Procedures in Periodontal Therapy, Dr. Lewis Fox and Associates. May 23-27, 1960.

For further information write to Director of Post-Graduate Studies, Department of Stomatology, Massachusetts Memorial Hospitals, 750 Harrison Avenue, Boston 18, Massachusetts.

EMORY UNIVERSITY

Emory University School of Dentistry will offer a course in clinical periodontia and oral pathology May 9-13, 1960. The fee for the course will be $150. For further information write to Dr. Ernest L. Banks, Director of Postgraduate Education, Emory University, Georgia.

BAYLOR UNIVERSITY COLLEGE OF DENTISTRY

The division of graduate studies, Baylor University College of Dentistry, Dallas, Texas, announces the following advanced courses of study in Periodontology, beginning in September, 1960:

1. A nine-month course of postgraduate study leading to the degree of Bachelor of Science in Dentistry. This course consists approximately half of advanced study in basic science and half of clinical study.

2. A twenty-month course (two regular school terms and one summer session) of graduate study leading to the degree of Master of Science in Dentistry. This consists essentially of the B.S.D. course above, followed by a summer term of clinical experience and then a second full nine-month term of advanced study in theory and clinical practice. A thesis is required.

Baylor has a modern school plant fully equipped for undergraduate and graduate study and completely air-conditioned. Every opportunity is offered the graduate student to prepare himself for the practice of Periodontology. For further information, write to the Registrar, Baylor University College of Dentistry, 800 Hall Street, Dallas 10, Texas.

Letter to the Editor . . . .

The Editor

I wish to comment on the diagnosis presented by Dr. John I. Ingle in his article: Papillon-Lefevre Syndrome: Precocious Periodontosis with Associated Epidermal Lesions, published in the July 1959 issue of the Journal.

The case was that of a boy, age 14, in which there had been an extreme labial movement of the upper right central incisor, the tooth having assumed an almost horizontal position. There was no corresponding movement of the left central incisor. Photographs and roentgenograms show no abnormality of gingivae or bone around the other upper incisors. Dr. Ingle calls this a case of periodontosis and links it with keratotic epidermal lesions on the hands, feet and arms. In support of his diagnosis he calls attention to the incipient bone resorption mesial to the lower first molars, also pocket formation—moderate on the first molars and slight on the lower central incisors.

I do not agree that the condition involving the upper right central incisor is a true periodontosis, even though the lesions mesial to the lower first molars suggests the presence of that condition in the mouth. It is difficult to conceive that a true periodontosis would involve only one central incisor. Also, in cases of juvenile periodontosis, as shown heretofore, the lower incisors are consistently affected more seriously than the upper incisors; also the first molars are more severely affected in relation to the incisors than in this case.

I believe that in this case the cause of the extrusion of the one upper central incisor is due to accidental trauma. No mention of this was made in the history and it is quite possible that for an athletically inclined boy, as in this instance, a blow to the tooth received during a game or vigorous play, might well have been disregarded at the time it occurred.

No mention was made of a pulp test, hence the matter of pulp vitality remains a question. The roentgenogram shows apical root resorption and slight periapical rarefaction. A blow such as I envision might well have caused pulp damage through rupture of the blood vessels at the apical foramen, resulting in loss of vitality without abscess formation but followed by the root resorption; this in itself may have initiated an extrusive action.

Further, the damage to the supporting tissues may have resulted in the formation of granulomatous tissue lingually to the root, with labial movement resulting from the expansion of that tissue.

I acknowledge of course the presence of an abnormal systemic condition and no not doubt that it may have some dental effect, hardly that, however, shown in the illustration. It will be interesting to follow this case and see what happens to the other teeth. Dr. Ingle is to be congratulated for bringing this case to our attention. I regret I cannot concur with his diagnosis regarding the upper right incisor.

John Oppie McCall, D.D.S.
944 Park Avenue
New York 28, N. Y.